University of Alberta

Genomic and Metabolic Mechanisms Leading to Variation in Feed Intake, Feed Efficiency, and Behaviour of Beef Cattle

BY

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ABSTRACT

The cost of feeding beef cattle accounts for more than 70% of the total variable cost in most production systems. Feed efficiency of beef cattle is therefore an economically relevant trait, which impacts the profitability and global competitiveness of the beef industry. Studies were conducted to characterize the metabolic and genomic mechanisms leading to variations in beef cattle feed efficiency. We employed the state-of-the-art radio-frequency-based GrowSafe® system to record the individual feed intake and behaviour of 464 Composite cattle over three years using the University of Alberta's experimental cattle population. Genetic and phenotypic parameter estimates for feed intake, feed efficiency, feeding behaviour, and temperament in beef cattle, and their genetic and phenotypic relationships with measures of growth and carcass merit are reported. It is demonstrated that differences among animals in heat production, digestibility, methane production, feeding behaviour, and energy retention may be responsible for a major part of the variation among animals in feed efficiency. It is further shown that the concentration of serum leptin in beef cattle is heritable, and there is moderate to high genetic and phenotypic correlations between serum leptin and carcass merit of beef cattle. However, correlations of bovine serum leptin with feed intake and feed efficiency were either low or not different from zero. The study reports associations of polymorphisms in the bovine leptin gene and its promoter with measures of growth, feed intake, feed efficiency, and carcass merit. Finally, the study reports a *Bos taurus* autosomal genome scan to locate quantitative trait loci for feed intake, feed efficiency, feeding behaviour, and temperament using genotypes from 455 genetic markers across 16 paternal half-sib families. The results of this study demonstrate ample opportunities for genetic improvement of beef cattle feed efficiency using metabolic- and genomics-based technologies.

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LIST OF ABBREVIATIONS

ADF	Acid detergent fiber
ADE	Apparent digestible energy
ADG	Averaged daily gain
AGRP	Agouti-related protein
ALP	Amplicon length polymorphisms
ARMS	Amplification refractory mutation systems
α-MSH	Alpha melanocyte stimulating hormone
ASO	Allele specific oligonucleotide
BBM	Brush border membrane
BLAD	Bovine leukocyte adhesion deficiency
BMR	Basal metabolic rate
BTA	*Bos taurus* autosome
BW	Body weight
CART	Cocaine and amphetamine regulated transcript
CCK	Cholecystokinin
CF	Crude fiber
CGF	Carcass grade fat
CIM	Composite interval mapping
CLMA	Carcass Longissimus muscle area
CMAR	Carcass marbling score
CNS	Central nervous system
CP	Crude protein
CRF	Corticotrophin releasing factor
CWT	Carcass weight
DE	Digestible energy
DGGE	Denaturing gradient gel electrophoresis
DM	Dry matter
DMI	Dry matter intake
EE	Ether extractives
EBV	Estimated breeding values

EPD	Estimated progeny difference
ERT	Economically relevant traits
EST	Expressed sequence tags
FCR	Feed conversion ratio
FD	Feeding duration
FE	Fecal energy
FF	Feeding frequency
FHP	Fasting heat production
FS	Flight speed
GE	Gross energy
GFE	Gross feed efficiency
GH	Growth hormone
GHRH	Growth hormone releasing hormone
GIP	Gastric-inhibitory polypeptide
GIT	Gastrointestinal tract
GLP	Glucagon-like peptide
HD	Head down time
HDL	High-density lipoprotein
HIF	Heat increment of feeding
HIm	Heat increment for maintenance
HIp	Heat increment for production
HP	Heat production
HWE	Hardy-Weinberg equilibrium
IBD	Identical-by-descent
IDL	Intermediate-density lipoproteins
IGF	Insulin-like growth factors
IGFBP	IGF binding proteins
LCAT	Lecithin cholesterol acyltransferase
LD	Linkage disequilibrium
LINES	Long interspersed nuclear elements
LMY	Lean meat yield
LPL	Lipoprotein lipase

MALDITOF	Matrix-assisted laser desorption/ionization time-of-flight mass spectrometry
MAS	Marker assisted selection
MCH	Melanin concentrating hormone
MCMC	Markov Chain Monte Carlo
ME	Metabolizable energy
MIM	Multiple interval mapping
ML	Maximum likelihood
MR	Maintenance requirement
MWT	Metabolic body size
NDF	Neutral detergent fiber
NE	Net energy
NEm	Net energy for maintenance
NEp	Net energy for production
NFE	Net feed efficiency
NFE	Nitrogen free extractives
NPY	Neuropeptide Y
OM	Organic matter
PDV	Portal drained viscera
PEG	Partial efficiency of growth
PIT1	Pituitary-specific transcription factor
POMC	Pro-opiomelanocortin
QG	Quality grade
QTL	Quantitative trait loci
QTN	Quantitative trait nucleotide
RAPD	Random amplification of polymorphic DNA
REML	Restricted error maximum likelihood
RFI	Residual feed intake
RFIg	Genetic residual feed intake
RFIp	Phenotypic residual feed intake
RFLP	Restriction fragment length polymorphisms
RLGS	Restriction landmark genome scanning

SAP	Specifically amplified polymorphisms
SINE	Short interspersed nuclear elements
SNP	Single nucleotide polymorphisms
SSCP	Single stranded conformational polymorphisms
SSRP	Simple sequence repeated polymorphisms (Microsatellite)
STDMI	Standardized DMI
STP	Standard temperature and pressure
STS	Sequence tagged sites
TDN	Total digestible nutrients
TNF	Tumor necrosis factor
UBF	Ultrasound backfat
UE	Urine energy
ULMA	Ultrasound longissimus muscle area
UMAR	Marbling score
VFA	Volatile fatty acids
VIP	Vasoactive intestinal polypeptide
VLDL	Very low-density lipoproteins
VNTR	Variable number of tandem repeats (Minisatellites)
YG	Yield grade

CHAPTER 1

General Introduction

1.1 Introduction

The cost of feeding is the single largest variable cost in beef production systems, accounting for approximately 70% of the total production cost (Perry and Cecava, 1995). Generally, about 70-75% of the total dietary energy consumed in a beef production system is used for maintenance (Ferrell and Jenkins 1984; NRC 1996). This means higher beef production costs, especially in large-sized breeding animals due to presumably higher maintenance energy needs, lower overall production system efficiency, and therefore lower profits. Indeed, compared to swine and poultry, which are able to convert about 14 and 22%, respectively, of the total energy intake into lean tissue depostion, only 5% of the total energy intake in beef cattle is converted into deposited protein. Improvements in the efficiency of feed utilization by beef cattle would therefore lead to better economic returns from both beef cattle breeding operations and feedlots (Gibb and McAllister, 1999; Liu et al., 2000; Herd et al., 2003). According to Johnson et al. (2003), the reason for the lack of change in beef cattle energetic efficiency, despite several years of intensive production include the lack of a consistent selection goal, loose and inconsistent definitions of efficiency, concentration on output traits, and emphasis on population differences rather than individual variation.

Efficient beef cattle production involves a complex summation of appropriate levels of available feed inputs and product outputs over a range of different production systems involving animals at different developmental stages. Thus, several indices have been proposed for determining the energetic efficiency of beef production, as comprehensively reviewed by Archer et al. (1999). These, among others, include feed conversion ratio (**FCR**), maintenance efficiency, partial efficiency of growth (**PEG**), cow-calf efficiency, and residual feed intake (**RFI**). Two other indices are relative growth rate (growth relative to instantaneous body size) and Kleiber ratio (weight gain per unit metabolic body size).

Traditionally, feed efficiency has been expressed in terms of FCR, or its inverse (gross feed efficiency, **GFE**). This is usually measured as the ratio of feed consumed to gain in weight. It somehow reflects the efficiency of use of the energy consumed for maintenance and growth and captures the relationship between input of feed and output of product (Herd et al., 2003). Though the concept of FCR has been in existence for many years, it is difficult to improve through direct selection because its genetic correlation with growth rate implies that selection for it may generally lead to an increase in body weight (**BW**) and subsequently feed intake, which is not always desirable (Gunsett, 1984; Archer et al., 1999; Crews, 2005). On the other hand, several studies in different species have demonstrated considerable phenotypic and genetic variation among individual animals in feed intake above and below the predicted requirements for maintenance and growth (Foster et al., 1983; Luiting and Urff, 1991; Archer et al., 1998; Archer et al., 1999). This variation in intake is usually measured as RFI, and was first proposed for use in cattle by Koch et al. (1963).

Sometimes referred to as net feed intake (**NFI**) or net feed efficiency (**NFE**), RFI relates to the variation in feed consumption among animals beyond that related to differences in growth rate and BW (Arthur et al., 1998). Residual feed intake has been shown to have greater potential as an index of energetic efficiency for beef cattle in terms of reduction in feed requirements with no compromise in growth performance of growing animals or increase in cow size when appropriately applied for selection (Liu et al., 2000; Herd and Bishop, 2000; Arthur et al., 2001a).

Several reports in the literature have highlighted the existence of considerable genetic and phenotypic variation among individual animals in RFI (Arthur et al., 1997; Archer et al., 1998; Herd and Bishop, 2000; Liu et al., 2000; Arthur et al., 2001a, b; Schenkel et al., 2004). Additionally, the heritability of RFI in growing cattle has been shown to be generally significantly different from zero, and often moderate (*ca.* 0.16 – 0.45, Herd et al., 2003; Crews, 2005). Results from studies reviewed by these authors generally show that RFI may be phenotypically independent of measures of production and maintenance and other measures of energy accretion, with the exception of body fatness, which shows a small correlation with RFI, although this result is not universally replicated across studies. More efficient beef cattle consume less feed than may be predicted from BW and growth rate over a certain period, and therefore have negative RFI.

From the above discussion of available research information, it is undeniable that genetic variation in feed intake and feed efficiency exists in beef cattle. What remains largely unknown however, are the biological and molecular mechanisms contributing to the variation in intake of animals of similar BW and growth rate. Several known biological mechanisms related to dietary energy transformations in the body may be proposed. These may include differences in the hormonal and enzymatic regulation of intake (Woods et al., 1998; Forbes, 2000), differences in energy losses during ingestion, digestion and metabolism (Luiting et al., 1994; Forbes, 2000; Reynolds, 2002; Basarab et al., 2003), efficiency of absorption and transport of nutrients (Zhao et al., 1998; Chen et al., 1999; Russell and Gahr, 2000; Mathews, 2000; Drackley, 2000), as well as differences in the rates and costs associated with energy accretion and overall turnover in muscle and adipose tissues (Reeds, 1989; Rathmacher, 2000; Reynolds, 2002).

Suggestive estimates following divergent selection for RFI indicated that the proportionate contribution to RFI from certain of the potential mechanisms were energy retained in protein and fat (5%), digestion (10 – 14%), feeding patterns (2 – 5%), heat increment of feeding (9%), level of activity (10%), protein turnover and tissue metabolism (37%), and about 27% associated with poorly characterized mechanisms such as ion transport and other substrate cycles in the body (Herd et al., 2004; Richardson et al., 2004). The present challenge lies not only in the need to establish the biological and biochemical bases for differences in feed efficiency between animals, but also in determining the genetic and biological bases for differences among animals at the molecular level. It is equally critical that we be able to translate fundamental knowledge gained through these important undertakings to functional understanding that can be applied to differences in the whole animal.

In the past, the collection of feed intake data for the accurate estimation of feed efficiency using relatively large numbers of beef cattle was almost impossible due to several difficulties associated with cost and accuracy. Recently, the availability of electronic feeding devices such as the radio-frequency based Growsafe System® (Basarab et al., 2003) have allowed the collection of feed intake and performance data on individual animals in a relatively large group setting. The University of Alberta experimental beef cattle population at the Kinsella Research Station provides an outstanding opportunity for designing specific crosses among cattle that will enable the measurement and characterization of the genetic and phenotypic variation in beef cattle feed efficiency. Data obtained from such studies have made it possible to characterize the extent of the phenotypic and genetic variation in feed intake, performance, and energetic efficiency, and to determine how these traits are related to measures of carcass merit of different beef cattle populations (Herd and Bishop, 2000; Liu et al., 2000; Arthur et al., 2001a; Basarab et al., 2003; Nkrumah et al., 2004).

Currently, it is possible to evaluate the physiological and metabolic basis of variation in feed efficiency with potential applications to reductions in manure and methane production. Classical techniques in ruminant nutritional energetics such as digestibility trials and metabolic studies may be employed to quantify differences between animals in the partitioning of dietary energy into faecal, urinary, methane, heat, and retained or product energy. Important energetics research tools such as indirect respiration calorimetry and comparative slaughter techniques may also be invaluable in such studies to establish the tissue and biochemical origins of heat production or energy expenditures (Johnson et al., 2003). Facilities at the University of Alberta Metabolic and Environmental Research Center present an excellent opportunity to undertake such studies on the whole animal at reasonable costs and efficiency.

Ultimately, the resulting phenotypic information collected using automated feed intake monitoring systems could be employed to dissect the molecular architecture of several economically relevant, but complex traits (**ERT**) in beef cattle. Molecular techniques can be employed to detect and map the chromosomal locations of genes contributing to variation in growth, feed intake, energetic efficiency, feeding bahviour, and carcass merit. Several molecular tools and approaches, as well as statistical and computational techniques, are available that can be employed to quantify the number(s), location(s) and effect(s) of quantitative trait loci (**QTL**) through the use of genotypic information from genetic markers that are spaced along chromosomes in the genome. The University of Alberta Bovine Genomics Laboratory houses facilities for high-throughput genotyping of genetic markers making it possible to conduct whole-genome QTL mapping studies. A QTL is defined as the chromosomal location of individual or groups of genes, of unknown primary function, that show(s) significant association with a complex trait of interest (Lander and Kruglyak, 1995). In beef cattle, QTL have been detected for disease tolerance (Hanotte et al., 2003), fertility and reproductive performance (Kirkpatrick et al., 2000), body conformation (Grobet et al., 1998), birth weight and growth performance (Davis et al., 1998; Casas et al., 2003; Li et al., 2002; Kim et al., 2003), and carcass and meat quality (Keele et al., 1999; Casas et al., 2000; MacNeil and Grosz, 2002; Casas et al., 2003; Kim et al., 2003; Moore et al., 2003; Li et al., 2004).

Finally, it is possible to search for associations between polymorphisms in specific candidate genes and measures of variation in feed intake, feed efficiency and feeding behaviour. A candidate gene may be selected based on previously known biochemical or physiological information or may be chosen because it maps to or close to the location of a QTL (positional candidate gene). Prominent among these candidates are genes shown to affect feed intake, behaviour, energy balance, and body composition, such as the appetite regulating gene leptin. Several polymorphisms in candidate genes have been shown to be associated with economically relevant traits in cattle (Chrenek et al., 1998; Barendse et al., 2001; Ge et al., 2001; Grisart et al., 2002; Buchanan et al., 2002; Moore et al., 2003; Li et al., 2004; Nkrumah et al., 2005).

1.2 Research hypotheses

The following research hypotheses were tested in the present research project:

1.2.1 Genetic and phenotypic variation in residual feed intake and other measures of energetic efficiency exist in the experimental beef cattle population, independent of BW, growth rate, or carcass merit.

1.2.2 Measures of feedlot energetic efficiency, performance and feeding behaviour are correlated with the metabolic rate, methane production and energy partitioning of beef cattle.

1.2.3 Genetic and phenotypic variation in feeding behaviour and temperament exist in beef cattle and are associated with BW, feed intake, feed efficiency, and carcass merit.

1.2.4 Circulating leptin levels in beef cattle are genetically and phenotypically related with BW, feed intake, measures of feed efficiency, and carcass merit.

1.2.5 Polymorphisms in the bovine leptin gene are associated with BW, feed intake, measures of feed efficiency, and carcass merit.

1.2.6 There are quantitative trait loci (QTL) for feed intake, feed efficiency and feeding behaviour traits segregating in the experimental cattle population.

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CHAPTER 2

Literature Review

2.1 Bovine nutritional energetics

The purpose of this section is to attempt to provide a brief overview of the general concepts and principles in ruminant nutritional energetics. It is not aimed at the more experienced reader in ruminant nutrition or animal science. It is a well-recognized fact that the determination of the nutrient requirements of farm animals is an essential pre-requisite for optimizing feed utilization, which eventually leads to increased livestock productivity, profitability, and conservation of feed supplies. Despite the role of the rumen in supplying some of the nutrient needs of cattle, like all farm animals, beef cattle require a source of nitrogen in the form of essential amino acids, fat in the form of essential fatty acids, essential mineral elements, a source of energy, and some of the fat- and water-soluble vitamins (NRC, 1984; Taylor et al., 1986). The amount of energy allowed in the diet is generally based on the basal metabolic rate (**BMR**) or resting energy requirement of the animal. The BMR of an animal is the amount of energy used by a fasted animal resting in a thermoneutral environment.

On the other hand, the composition of the diet in terms of the individual nutrients is based on the maintenance requirement (**MR**), which is the amount of energy required to maintain the animal's BW (no gain or loss) at moderate physical activity (NRC, 1987). The MR is usually equivalent to twice the BMR of the animal (Church and Pond, 1988). A number of abbreviations, as contained in Nutritional Energetics of Domestic Animals and Glossary of Energy Terms (NRC, 1981), are often used to describe energy fractions in the animal system. Routinely, the dry feed (moisture-free sample) of an animal is expressed as the dry matter (**DM**) or the organic matter (**OM**) content. The DM can be subjected to proximate analysis to yield many different fractions of feed components. The major components include crude protein (**CP**) (made up of different types of protein and non-protein nitrogen), ether extractives (**EE**) (made up of true fats, fatty acid esters, compound lipids, and fat-soluble vitamins or pro-vitamins), and ash (residue remaining of sample after all combustible materials have been completely oxidized in a furnace at 500-600°C).

Others are crude fiber (**CF**) (made up of cellulose, hemicellulose and lignin), and nitrogen free extractives (**NFE**) (made up of some readily available carbohydrates, but also hemicellulose and lignin). The NFE fraction is obtained by difference (i.e. when the sum of the weights of moisture, EE, CP, ash, and CF are subtracted from the weight of the original sample) (Van Soest, 1982). Samples of feed can also be subjected to detergent extraction to produce certain nutrient extracts. The major ones are neutral detergent extraction to produce neutral detergent fiber (**NDF**) and acid detergent extraction to produce acid detergent fiber (**ADF**). Neutral detergent (usually sodium laural sulfate) extracts lipids, sugars, organic acids, other water-soluble materials, pectin, non-protein nitrogen compounds, soluble proteins, some silica, and tannins; it leaves a residue of celluloses, hemicelluloses, and lignin. On the other hand, acid detergent (usually cetyl trimethylammonium bromide with H_2SO_4) extracts primarily hemicelluloses and cell wall proteins, residues of celluloses, lignin and lignified nitrogen (indigestible nitrogen), cutin, silica and some pectins (Van Soest, 1982 and 1994).

The amount of feed offered to an animal is usually stated on the basis of DM or OM, and all the components (energy, protein, mineral, vitamins, etc.) are expressed as a proportion of DM. The overall energy content of the feed consumed by an animal is expressed as the gross energy (**GE**). The GE is measured as the heat of combustion liberated after complete oxidation of a sample of the feed in a bomb calorimeter. Generally, GE is expressed in kilocalories per gram of sample (kcal/g) or mega calories per kilogram (Mcal/kg); one kcal is equivalent to 4.184 kilojoules of energy in SI units. Generally, a gram of carbohydrate, fat, and protein, respectively, would yield 4.0 kcal, 9.0 kcal and 4.0 kcal of energy (Church and Pond, 1988; NRC, 2000). Beef cattle would rarely utilize the total energy in the feed consumed due to the amount voided as faeces. The proportion of the energy in the diet utilized by the animal has been termed apparent digestible energy (**ADE**) and it is determined by subtracting the amount of faecal energy (**FE**) (also obtained by bomb calorimetry) from GE. However, the energy in the faeces also contains energy from rumen microbes, intestinal secretions and sloughed intestinal lining (ARC, 1980). Thus, ADE may be corrected for these energy sources to obtain the true digestible energy (**DE**).

Another terminology, total digestible nutrients (**TDN**) is sometimes loosely used to mean DE. In actual terms, TDN equals the sum of digestible CP, digestible CF, digestible NFE and 2.25 times digestible EE. To make TDN more comparable to DE, the amount of CP needs to be multiplied by a factor of 1.25 (NRC, 1981). Digestible energy is further refined to produce metabolizable energy (**ME**), after correcting for energy losses in the form of combustible gases of ruminal microbial fermentation (mainly methane, but also carbon dioxide, hydrogen, acetone, ethane, hydrogen sulfide, nitrogen, and oxygen) and urine energy (**UE**) from non-utilized, absorbed compounds and end products of metabolic processes (Church and Pond, 1988; NRC, 1996). According to NRC (1996), ME in beef cattle can be calculated from DE with the equation ME = DE x 0.82. However, for feedlot cattle on high grain diets that may also contain vegetable fats or ionophores such as monensin, the proportion of intake energy lost through urine and methane are considerably lower than for high roughage diets (Van der Honing and Steg, 1990) and the ratio of DE to ME may be considerably higher. The practice of adding vegetable oils and ionophores to high grain feedlot diets to reduce extreme cases of bloat have especially been shown to depress rumen methanogenesis considerably (Mathison, 1997; McGinn et al., 2004).

Finally, ME can be converted to net energy (**NE**) by eliminating energy losses in the form of heat production (**HP**) or heat increment of feeding and metabolism (**HIF**). Cattle produce heat during nutrient ingestion, microbial fermentation, absorption, assimilation, metabolism and formation of waste products (NRC, 2000). Heat increment is generally partitioned into heat increment for maintenance (**HI_m**) and heat increment for production (**HI_p**). Net energy can also be partitioned into net energy for maintenance (**NE_m**) and net energy for production (**NE_p**) (Fan et al., 1995; NRC, 1996). Net energy for maintenance of growing bulls, steers and heifers has been proposed to be estimable from BW as $NE_m = 0.077\ BW^{0.75}$, where BW is the average empty body weight (kg) (Garret, 1980). Similarly, the net energy for gain (NE_g) can be estimated as $NE_g = 0.0493\ (BW)^{0.75}$ x $(LWG)^{1.097}$/days on test, where BW = average body weight and LWG = total live weight gain (Fan et al., 1995). Generally, such requirements may be adjusted for age, growth, breed, reproductive status, and environmental factors. Maintenance energy needs may also be determined exponentially on the basis of metabolic body size or weight ($BW^{0.75}$).

Beef cattle, like all farm animals, are fed based on the physiological state and the expected level of production. Energy requirements for maintenance are positively associated with the level of production achievable by the animal (Ferrell and Jenkins, 1984). It is therefore important to concentrate on maintenance requirements before evaluating potential production level and costs when studying differences in beef cattle productivity and economic efficiency. Indeed, the metabolic capacity required to produce a large amount of meat increases the overall energy requirement for maintenance (Jenkins et al., 1986; DiConstanzo et al., 1990).

2.2 General factors affecting feed intake in beef cattle

There are numerous factors that can interfere with the concordance between nutrient requirements and feed intake by beef cattle. These factors are significant to the determination of feed efficiency since the factors controlling feed intake and production cost are interrelated. As an illustration, it has been observed that the maintenance requirements of animals eating less and gaining at a slower rate represents a much greater proportion of the total feed required than for animals gaining at faster rates (McCullough, 1973). In the above example, maintenance accounted for 75%, 59%, and 48% of total energy for cattle gaining at 0.45, 0.91, and 1.36 kg/d, respectively. The general factors that affect feed intake in beef cattle may broadly be grouped under animal factors, feed or nutritional factors, environmental factors, management or artificial factors and physiological or metabolic factors (Church and Pond, 1988; NRC, 2000). There are however, complex interrelationships between the above classes of factors.

2.2.1 Individual animal factors

The most important animal factors that affect feed intake of beef cattle, with regards to this study, are breed or genetic differences between animals. Several reports in the literature have indicated considerable genetic differences between beef cattle in terms of feed intake. For instance, Garrett (1971) reported that Holstein steers required 23% more feed to maintain body weight than Hereford steers. Also, Ferrell and Jenkins (1985) indicated that feed required for weight or energy stasis in young bulls and heifers was greater in Simmental than in Hereford. Other similar reports include Blaxter and Wainman (1966), Solis et al. (1988), Jenkins et al. (1991), and Laurenz et al. (1991). Significant differences among genders (bulls, heifers, and steers) in feed intake and maintenance requirements have also been reported (Geay et al., 1980). CSIRO (1990) indicated that the maintenance requirements for bulls are 15% higher than that for steers or heifers, even if they are of the same genotype. Additionally, Ferrell and Jenkins (1985) indicated that differences between heifers (69.3 kcal/$BW^{0.75}$/day) and bulls (70.4 kcal/$BW^{0.75}$/day) in fasting heat production (FHP) accounted for sex differences in feed intake.

The age of beef cattle at the time of introduction to a feed type also influences feed intake. Generally, older animals consume more feed per unit of body weight than younger animals (Graham et al., 1974; NRC, 1984; Tyrell and Reynolds, 1988; Young et al., 1989; CSIRO, 1990). It has been shown that body weight or size and composition, especially percentage of body fat affects the level of intake in cattle and other farm animals (Noblet et al., 1998). According to Church and Pond (1988), beef cattle introduced to feedlots with approximately 10% body fat consume about 2.5-2.75% of BW per day, but nearing finishing, when body fat is generally 25-30%, beef cattle usually consume up to 2.2% of BW in a day. Forbes (2000) and Houseknecht et al. (1998) explain that leptin from mammalian adipose tissues is known to play a negative feedback role in feed intake regulation. The physiological state (gestating, lactating or dry) and level of production of cattle also influence the amount of feed consumed (Moe et al., 1970; Minson, 1990). Finally, the previous plane of nutrition or compensatory growth affects level of feed intake by cattle (Anderson, 1980; Carstens et al., 1991). Evidence of increased intake by cattle experiencing compensatory growth has also been provided by NRC (1987) and Ryan et al. (1993).

2.2.2 Feed or nutritional factors

The quantity of feed available to beef cattle (NRC, 1987; Minson, 1990), the degree of processing (NRC, 1987; Galyean and Goetsch, 1993), and the palatability (Church and Pond, 1988), as well as textural and sensory characteristics like sight, taste, odour, and coarseness or fineness (Campion and Leek, 1997) are some of the important feed factors that influence the level of feed consumption in farm animals, including beef cattle. The most important nutritional factor that has been shown to greatly affect feed intake is the nutrient composition of the feed, especially the caloric density (Church and Pond, 1988; Forbes, 2000). Also important is the composition of essential amino acids, essential fatty acids, essential minerals, and certain water- and fat-soluble vitamins (Kutlu and Forbes, 1993; Forbes, 1995).

For example, Galyean and Goetsch (1993) believe that a dietary nutrient deficiency, particularly protein, can decrease feed intake. According to these authors, nitrogen deficiency is common with low-nitrogen, high-fiber forage, and the provision of supplemental nitrogen often increases dry matter intake substantially. According to NRC (1987), forage intake responses to protein are most typical when forage crude protein content is less than 6 to 8 percent. It has also been shown that the level of dietary fibre has a significant, but variable, influence on the energy density of feed consumed and hence the level of intake (Reynolds, 1996; Yan et al., 1997). Generally, with high fiber diets, there is increased dry matter intake to achieve similar levels of ME intake leading to greater gut fill, greater work during rumination, and greater energy usage for fermentation (Agnew and Yan, 2000).

2.2.3 *Environmental factors*

The major environmental factor that influences beef cattle feed intake is ambient temperature. In general, feed intake increases when temperature falls below the thermoneutral zone, as the animal must consume more feed to generate enough heat to regulate body temperature. On the other hand, feed intake decreases when temperature is above the thermoneutral zone, since increased intake under such conditions culminates in heat stress in the animal. However, these general patterns of response may vary depending on dietary factors, local conditions, acclimatization, and environmental susceptibility of the animal (Kennedy et al., 1986; Minton, 1986; Young et al., 1989).

Seasonal variations in day length, otherwise known as photoperiodism, though has less fully understood effects, has been suggested as being very important in controlling intake in beef cattle (NRC, 1987; Hicks et al., 1990). For instance, in Danish Black and

White heifers, bulls and steers, daily dry matter intake has been shown to increase by 0.32% per hour increase in day length (Ingvartsen et al., 1992). Adverse environmental conditions such as diseases and pests (Church and Pond, 1988), trauma and noise, precipitation, floods, winds, snow, mud, and bush fires may all affect feed intake, sometimes through the invocation of a stress response in the animal (Fox et al., 1988; Delfino and Mathison, 1991).

2.2.4 Management or artificial factors

Factors affecting intake such as forage availability and quality (NRC, 1987; McCollum et al., 1992) and dietary nutrient composition (Galyean and Goetsch, 1993), as discussed under feed factors, are to some extent, management decisions. Other management factors like the use of growth-promoting implants tend to increase feed intake. For instance, it has been indicated that beef steers with estradiol benzoate/or progesterone implants increased their dry matter intake from 4 to 16% (Rumsey et al., 1992). On the other hand, lasalocid, an ionophore also approved for use in beef cattle, seems to have limited effects on beef cattle feed intake. Fox et al., (1988) suggested that feed intake is decreased by 2 % regardless of dietary concentration of lasalocid. Galyean et al. (1992) however reported a 4% increase in feed intake of a diet containing 33mg lasalocid/kg diet compared to a nonionophore control diet. Also, 33 mg and 22 mg/kg diet of the ionophore monensin decreased feed intake by 10 and 6 percent, respectively, (Fox et al., 1988). Galyean et al. (1992) noted a 4 % decrease in feed intake when cattle were fed 31 mg monensin/kg dietary dry matter.

Other important factors may include the time an animal spends looking for feed and the amount of physical activity involved in grazing (Adam et al., 1984). Finally, there are other physiological processes and substances such as enzymes, hormones, and other digestive juices, as well as some tissues, organs and systems, that may have an influential regulation (directly or indirectly) on beef cattle feed intake, but these are discussed in later sections of this review.

2.3 Measurement of feed intake and feed efficiency in beef cattle

Providing feed to cattle is the single largest expense in most commercial beef production enterprises (Perry and Cecava, 1995). Thus much research emphasis is now being placed on reducing intake but improving efficiency of feed use in order to lower input costs. To include feed intake and efficiency data in making selection decisions, appropriate and accurate measures of these traits are required. Measurement of individual animal feed intake in cattle is usually difficult, slow and very expensive. As a result, any information obtained from feed consumption and growth performance of cattle is of a very high value for use in research and breeding programs (Arthur et al., 2004). There is very little information available on the maximum and minimum intake measurement durations that would lead to substantial cost reductions without compromising the accuracy of experimental results (Archer et al., 1997). Recent advances in computing and electronics technology have now made the centralized estimation of feed consumption and animal performance relatively easy through the use of automated individual feeding systems (Basarab et al., 2003; Arthur et al., 2004). These facilities do not only have the capacity for the quick and efficient measurement of feed intake and animal performance but also allow relatively uniform environmental conditions to reduce the sources of variation in experimental results.

Intensified selection in beef cattle for faster growth rate over the years in an effort to increase profitability has resulted in an increase in the mature BW of animals, which in turn have higher maintenance energy requirements and a corresponding increase in feed consumption for production (DiCostanzo et al., 1990). Another problem that is associated with selection for higher growth rates is that the increase in mature BW is predominantly expressed in the parental stocks that become heavy and expensive to manage. This has led to an antagonistic relationship between the advantages of increased growth rate (as expressed in growing cattle) versus higher maintenance requirements and cost of maintaining the breeding population (Taylor et al. 1986; Liu et al., 2000).

According to Luiting et al. (1994), one-sided selection for higher production will automatically lead to animals, especially adult cows, with increased maintenance requirements, higher feed requirements, higher production costs, less efficient usage of feed resources, high heat production, higher CO_2 production, higher methane emission, higher manure production, and higher environmental pollution. Liu et al. (2000), therefore argue that traditional selection for faster growth alone may not necessarily result in efficient animals as the rate of gain is only moderately correlated with feed intake. Selection for rapid growth rate and bigger sizes may therefore increase revenue, but it may also result in increased production costs due to increased feed intake and increased waste outputs, and hence increased environmental pollution (Luiting et al., 1994).

There is therefore the need for strategies to lower feed inputs and waste outputs, not only in growing animals, but also in adult cattle, in order to optimize the overall production system efficiency. This can be achieved through the selection of animals with superior efficiencies in feed utilization (Arthur et al., 1998). Selection of animals with superior feed efficiencies requires an index for the estimation of efficiency in the whole production system. It is preferable that such an index be independent of the level of production or product quality. According to Archer et al. (1999), the overall efficiency of a beef production system is a complex biological trait and depends on the summation of the effects of many traits expressed in the breeding herd and the slaughter generation, and whose relative importance differ depending on the production system.

To obtain improvements in overall production system efficiency, the index (or indices) selected to measure efficiency must be correlated to production system feed efficiency, and must allow genetic improvements in feed efficiency that can be expressed in growing animals and in the cow herd. Several indices have been proposed for estimating feed efficiency in farm animals ranging from those that could be employed in growing animals to those employable in mature animals (Arthur et al., 1998). These indices have been fully reviewed by Archer et al. (1999) to include gross feed efficiency (or feed conversion ratio), maintenance efficiency, partial efficiency of growth, cow-calf efficiency, and net feed efficiency (estimated as net or residual feed intake). Two other indices are relative growth rate (growth relative to instantaneous size) and Kleiber ratio (weight gain per unit metabolic body size) (Archer et al., 1999). Though all the above indices relate to variation in feed efficiency, they may capture different sources of variation in the nutrient transformation process.

2.3.1 Feed conversion ratio

Feed conversion ratio (FCR), or its inverse (gross feed efficiency, GFE) may be defined as the ratio of output to inputs. In beef production, it may be defined as the weight of beef produced per unit weight of feed consumed over a certain defined time period (Brelin and Brannang, 1982). It may also be expressed on weight constant or maturity constant basis (Salmon et al., 1990). It is well known that selection based on FCR leads to increments in efficiency in beef cattle during the growth and finishing stage (Luiting et al., 1994), and has results similar to selection for growth rate. This makes it highly beneficial to the feedlot industry (Brelin and Brannang, 1982; Mrode et al., 1990).

However, according to Okine et al. (2001), since feed conversion ratios are highly correlated with growth, they are greatly confounded with growth rate and maturity patterns. In other words, favourable reductions in FCR are not necessarily correlated specifically to improvements in feed efficiency, but may only reflect selection for increased growth rate (Crews, 2005). In addition, FCR is defined as a fraction, and changes in either of the components of the ratio may result in a disproportionate amount of selection pressure being placed on the component of the ratio with the highest genetic variation (Gunsett, 1984). Thus, selection against FCR might have unfavorable effects on overall production system efficiency (Barlow, 1984).

Using FCR as an index of feed efficiency would therefore result in substantial increases in the feed requirement of the cow herd, which would then negate the gains made from the high growth rate of young animals and result in negative impacts on the overall production system efficiency (Andersen, 1978; Dickerson, 1978; Barlow, 1984). Archer et al. (1999) therefore concluded that, although some gains may be made in feedlots or in breeds used to provide terminal sires, or in nutritional studies involving similar genotypes through the use of FCR in selection, the associated increase in the mature sizes of cows in the breeding herd would increase the cost of feeding and compromise overall production system efficiency.

2.3.2 Maintenance efficiency

Estimates based on typical temperate beef production systems have indicated that maintenance energy requirements account for 65 – 70% of total beef production energy requirements (Montano-Bermudez et al., 1990; Liu et al., 2000). Depending on the production system, the cow herd has been shown to expend 60 – 85% of the total maintenance energy (Montano-Bermudez et al., 1990). This therefore makes the cost of maintaining the breeding cow a very important component factor in determining production system efficiency and profitability.

However, the use of maintenance efficiency as an index of feed efficiency is confounded by many difficulties in its estimation. In the first place, maintenance efficiency estimation requires that animals be held at a constant BW, which can take two years or more in beef cattle (Taylor et al., 1981). It is also known that other measures of maintenance such as fasting heat production reflect other factors instead of maintenance alone and are also affected by the growth of the animals prior to its measurement (Koong et al., 1982). This makes it immeasurable in growing animals and precludes maintenance efficiency as a possible index for estimating production system efficiency in cattle that can be used in genetic improvement programs.

2.3.3 Partial efficiency of growth

Partial efficiency of growth (**PEG**) is defined as the weight gain per feed intake less the animal's maintenance requirements (Archer et al., 1999). Though PEG has been shown to be highly correlated with feed intake and other measures of efficiency such as FCR and RFI (Arthur et al., 2001a; Nkrumah et al., 2004b), its measurement requires the estimation of maintenance requirements either from feeding standards tables or based on the rate of metabolism (ARC, 1980). However, estimation of maintenance requirements from feeding standards tables is based on the assumption that no variation exist among individual animals in the use of maintenance energy. This assumption has however been shown to be false (Archer et al., 1999). Though it could be estimated through metabolic studies, it requires constant liveweight of animals, just as in maintenance efficiency estimation. Partial efficiency may therefore not be very feasible as an index for measuring overall production system efficiency.

2.3.4 *Cow-calf efficiency*

This is a measure of the weight of calf weaned per unit weight of feed consumed by both the cow and its progeny, and it is calculated by measuring the feed intake of the cow and her progeny over a production cycle (from one weaning period to another) and comparing to the weight of calf weaned (Shuey et al., 1993; Jenkins and Ferrell, 1994). The index has a high likelihood of being correlated to beef production system feed efficiency in both biological and economic terms. The procedure however does not consider the feed consumption of the slaughter generation and that of breeding replacements (Jenkins and Ferrell, 1994). In addition, the index is difficult to use in practical situations and in genetic studies because its estimation requires considerable effort and expenditure. Also, the genes derived from the sire confound the genetic merits of cow/calf systems. Nevertheless, it could be used as an indicator of the level of phenotypic variation in production system feed efficiency in beef cattle.

2.3.5 *Residual feed intake*

Residual feed intake (**RFI**) is severally referred to as net feed intake or net feed efficiency and it refers to the variation in feed consumption between animals beyond that related to differences in growth rate and body weight (Arthur et al., 1998). Stated differently, RFI is the difference between an animal's actual feed consumption and its expected feed consumption for maintenance and growth over a specific test period (Arthur et al., 2001b). It is an index for comparing observed efficiency of feed utilization with predicted efficiency of feed utilization. Most published studies have computed RFI from a phenotypic regression of ADG and metabolic BW on feed intake. Being based on the difference between observed intake and predicted requirements, RFI may be forced to be phenotypically independent of growth and maturity patterns (Fan et al., 1995; Liu et al., 1998). In reality, if the ME intake equals ME required for maintenance and gain, then the predicted requirements of the animal are completely met by energy intake. A positive RFI therefore means that the animal's energy intake exceeds its predicted requirement and the animal is less efficient, but a negative RFI means that the animal either requires less energy than predicted or is eating less to produce the same weight gain (Okine et al., 2001).

According to Kennedy et al. (1993), since RFI is phenotypically independent of production, it may allow comparisons between individual animals differing in level of production during the measurement period. The latter authors have however made suggestions that RFI computed in this manner may not be genetically independent of production, thereby raising concerns over responses to selection over the long term. Since genotypic RFI may be genetically independent of production, it may be more reflective of underlying genetic differences between animals (Archer et al., 1999). However, Kennedy et al., (1993) also showed that, even though RFI computed from genetic regression may be genetically independent of production, there is very limited variation in genetic RFI between animals. Also, estimates of genotypic RFI are similar to restricted genetic selection indices, and selection on genetic RFI may be considered sub-optimal (Crews, 2005). Indeed, Arthur et al. (2001a, b) showed that genetic correlations between phenotypic RFI and production traits may be near to zero, making any potentially unfavourable correlated responses to selection for RFI very negligible.

Further studies are obviously required to establish the relationship between phenotypic and genetic RFI and to compare how these relate to other measures of production and product merit. Recent reports by Hoque and Oikawa (2004) and Hoque et al. (2005) indicate that the genetic and phenotypic correlations between phenotypic RFI and genetic RFI are greater than 0.90, making the two indices essentially the same. It is important for the industry to know that, as an index of selection to reduce feed intake, the application of RFI in cattle evaluations is going to have no unfavourable changes to body weight or growth rate. This is to ensure that the technology will be equally beneficial to both feedlot operations and breeding herd managers in terms of improvements in feed efficiency and reduction in feeding cost. This makes it a potentially acceptable index for measuring feed efficiency that could be used for genetic improvements to increase production system efficiency.

2.4 Genetic variation in feed intake and feed efficiency of beef cattle

The use of genetic evaluation procedures to identify animals with the highest genetic merit, which can be used as parents for future generations, is an important component of every animal breeding and selection program. In the case of feed efficiency the goal is to improve the genetic merit in the overall production system. The statistical procedures used to obtain such genetic parameter estimates are based on Henderson's mixed model equations (Henderson, 1984), and provide best linear unbiased predictions (**BLUP**) of genetic merit for individual animals in the form of estimated breeding values (**EBV**) or expected progeny differences (**EPD**). Not only has BLUP become the present standard for genetic prediction, but specialized software programs have been developed to efficiently implement the complex statistical procedures that are involved (e.g. Boldman et al., 1995; Gilmour, 1997). The genetic improvement approach requires the evaluation of both young and adult individuals within and across breeds based on the selected index such that any gains made from selection would be transmitted along the entire length of the production chain.

Evidence of genetic variation in beef cattle feed intake has been reported in several studies. Recent reports of heritability irect destimates for feed intake include 0.31 ± 0.08 (Herd and Bishop, 2000), 0.39 ± 0.03 to 0.48 ± 0.04 (Arthur et al., 2001a), 0.27 ± 0.06 (Robinson and Oddy, 2004), 0.44 ± 0.06 (Schenkel et al., 2004), and 0.34 ± 0.11 (Hoque et al., 2005). Evidence for genetic variation in indices of feed efficiency published for cattle up to the mid 1990s were summarized by Archer et al. (1999). There are a considerable number of reports in the literature indicating the existence of genetic variation in FCR in growing beef cattle of different breeds. Reported heritability estimates for FCR include 0.35 ± 0.24 (Brelin and Brannang, 1982), 0.33 ± 0.10 (Mrode et al., 1990), 0.26 (Bishop et al., 1991), 0.16 ± 0.14 (Fan et al., 1995), 0.31 ± 0.09 (Gengler et al., 1995), 0.20 to 0.27 (Arthur et al., 1998), 0.17 ± 0.09 (Herd and Bishop, 2000), and 0.29 ± 0.04 to 0.46 ± 0.04 (Arthur et al., 2001a, b).

The study by Arthur et al. (2001b) provided evidence for direct heritability estimates for PEG (0.39 ± 0.05), Kleiber ratio (0.31 ± 0.05), and relative growth rate (0.33 ± 0.04). Several studies in the early 1990s reported genetic parameter estimates for RFI in both growing and lactating cattle (Korver et al., 1991; van Arendonk et al., 1991; Jensen et al., 1992; Ngwerume and Mao, 1992). Several recent studies have also reported genetic variation in RFI in a range of beef cattle breeds. Heritability estimates for RFI in these studies are generally different from zero and include 0.16 ± 0.08 (Herd and Bishop, 2000), 0.39 ± 0.03 (Arthur et al., 2001a, b), 0.26 to 0.30 (Crews et al., 2003), 0.38 ± 0.07 (Schenkel et al., 2004), and 0.24 ± 0.11 (Hoque et al., 2005). Hoque et al. (2005) recently estimated the heritability of genotypic RFI to be 0.25 ± 0.10.

Details from the studies cited above indicate that feed intake and FCR are positively correlated with each other. The genetic correlation estimates of feed intake with growth and BW are positive, ranging from 0.25 to 0.80. On the other hand, genetic correlation estimates of FCR with growth rate and BW are negative, ranging from -0.20 to 0.90. The strong genetic association of FCR with growth rate and BW makes it difficult to determine whether the variations in gross efficiency between individuals simply represent the variations in growth (Liu et al., 2000). As a result, selection for FCR will make large-sized animals more efficient during the growing stage (in feedlots) but these animals may have higher maintenance requirements in the breeding herd, making cow-calf operations unprofitable (Archer et al., 1999).

The genetic and phenotypic correlation of phenotypic RFI with PEG, FCR, and feed intake are favourable, and it can be shown to be phenotypically independent of growth and BW. According to Kennedy et al. (1993), though phenotypic RFI may theoretically be phenotypically independent of maintenance and production, it may not be genetically independent of maintenance and production. These authors therefore suggested that, in growing animals, instead of using phenotypic regression, genetic regression should be used to derive residual feed consumption so that the resulting feed consumption is genetically independent of maintenance and production.

Studies by Archer et al. (1998) and Herd and Bishop (2000), as well as a recent study by Hoque et al. (2005) showed that the phenotypic RFI that was phenotypically independent of maintenance and production was very highly correlated (r = 0.97) with the genetic RFI that was genetically independent of maintenance and production. In addition, Hoque et al. (2005) showed that, the relationship of phenotypic RFI and genotypic RFI with growth rate and BW may not be different. The advantage of phenotypic RFI lies in the significantly higher level of genetic variation, compared to genotypic RFI (Crews, 2005). Further studies are obviously required to confirm the genetic relationship of different measures of RFI with indices of maintenance and production as well as carcass quality. In summary, considerable genetic improvement can be achieved by selecting for appropriate measures of feed efficiency without significantly changing the maintenance requirements and production levels of the animals involved. The challenge now is the need for further studies to uncover the biological and molecular mechanisms underlying such variations in beef cattle populations. This would allow the exploitation of such variations in breeding and selection programs to improve production system feed efficiency and reduce feeding costs in beef production.

2.5 Mechanisms contributing to variation in feed intake and efficiency

In section 1.1, suggested estimates from potential sources of variation in feed efficiency, as proposed by Herd et al. (2004) and Richardson et al. (2004) were presented. Biological mechanisms contributing to genetic variation in efficiency may broadly be classified under sources due to individual animal differences in rate of gain, BW, prolificacy, feed intake, digestion and absorption, metabolism (maintenance and growth metabolism), physical activity, and thermoregulation (Oddy and Herd, 2001; Johnson et al., 2003). Of these, rate of gain, BW, prolificacy, and to some extent, thermoregulation, have received most of the previous research attention, and will not be covered in this section.

2.5.1 Biological mechanisms regulating feed intake and energy balance

The amount of food consumed by an animal in a given time period and its processing and partitioning to meet various requirements of the animal forms an important part of animal growth and development. In section 2.2, the general factors that affect feed intake in cattle were discussed. Generally, two models have been proposed as forming the basis for food intake regulation in animals. There is a depletion-repletion model, which suggests the constant monitoring of some parameter of immediately available energy, with declining amounts triggering onset of feeding. Initiation of feeding therefore occurs when a threshold available energy value is reached and is terminated when levels are sufficiently replenished. An example of this is the classical glucostatic model hypothesis (Mayer and Thomas, 1967), which has both the brain and the liver monitoring and triggering or terminating intake as and when appropriate. Literature evidence however points towards the fact that, though body energy depletion and repletion correlate well with food intake, they are poorly related to energy expenditure and partitioning (Woods et al., 1998). The depletion-repletion model, though accurate in some respects, does not fully account for long-term stability of body energy stores by matching intake to expenditure.

The second model (lipostatic model) matches intake with amount of stored energy in the body and argues that signals proportional to the fat stores in the body are integrated with other intake regulatory mechanisms to control feed consumption. Neither is meal onset or termination tied to depletion nor replenishment of substrates respectively, but initiation and/or termination occur due to reasons such as environmental conditions, habits and learned associations, opportunity, social factors, time of the day, as well as signals generated in proportion to fat mass (Woods et al., 1998). Energy needs are therefore met by utilization of recently available calories and by drawing on stored energy at other times whilst the depletion of energy stores (adipose tissue) increases consumption through a primary increase in meal size. Frequent meals may be consumed when food or energy availability becomes severely depleted. The lipostatic model is widely supported by a wide range of published literature evidence. Under this model, therefore, feed intake and energy partitioning do not involve a single factor but are made up of a complex synthesis and the integration of the sum total of both external and internal stimuli. The external stimuli are made up of dietary and environmental cues and the internal physiological signals involve all controls as signaled by maintenance requirements and potential production (Baile and Della-Fera, 2001).

Evidence in the literature shows that regulation of feed intake and energy homeostasis can be divided into physiological pathways not directly involving the central nervous (**CNS**) system and pathways that directly originate or are triggered by CNS control. The non-CNS physiological pathways may include the effects of the gastro-intestinal tract (**GIT**) as well as other organs of the body such as the liver and the sensory organs. There is however no clear-cut distinction between which regulatory mechanisms are hormonal and non-hormonal since even signals which are not primarily of hormonal origin may be influenced by hormonal pathways. The CNS pathways involve those pathways that are under the direct influence of the hypothalamic, somatotropic, or adrenal axis (Woods et al., 1998; Baile and Della-Fera, 2001).

The role of the GIT in feed intake regulation in ruminants may be related to changes due to the degree of fill and the chemical composition of the digesta, which can be sensed by stretch receptors and chemoreceptors in the wall of the digestive tract (Forbes, 2000). In ruminant livestock, it is believed that GIT regulation of intake primarily occurs at the level of the rumen through a number of mechanisms. Limited capacity of the rumen to accommodate feed (despite its large size) due to the slow rate of digesta passage, the presence of sensory receptors on the rumen walls (especially on the anterior dorsal part that are sensitive to distension and can convey satiety signals to the hypothalamus through the vagus nerve), fibrosity of the feed consumed, particle size, and gut motility, among other factors, all participate in intake regulation through effects on rumen fill (Anil et al., 1993; Forbes, 2000).

Also, there are rumen epithelial receptors sensitive to the levels of volatile fatty acids (**VFAs**) in the rumen, and this signals to the central nervous system about whole-body energy status. For instance, a study with ram lambs shows that increased VFA concentrations in the rumen, among other things, have a great capacity for feed intake depression (Cole, 1991). There are also factors related to the chemical status, as well as the osmolarity of the GIT. Anil et al. (1993) studied the effect of infusion into the rumen of sodium acetate or propionate, and of distension (by means of an inflated balloon) on intake of hay or silage by fistulated lactating cows. The results showed depression of feed intake by all three treatments in the experiment. Some of the effects of this feed intake depression by substrate infusion has been shown to be related to the osmolarity of chemicals which influence intake through an osmoreception mechanism (Rooke, 1995), but others are related to GIT distension.

The GIT plays a major role in feed intake and energy-balance regulation through the effects exerted by several gastrointestinal hormones. The most common and essential GI hormones that are known to regulate digestive function of the GIT include gastrin, gastric-inhibitory polypeptide (**GIP**), secretin, cholecystokinin (**CCK**), glucagon-like peptides (**GLPs**), somatostatin, pancreatic polypeptide and vasoactive intestinal polypeptide (**VIP**) (Church and Pond, 1988). For instance, it has been shown that GIT administration of CCK causes a dose-dependent decrease in feed intake and induces satiety behaviour (Smith and Gibbs, 1984). These authors believe that CCK stimulates gut motility and subsequently ruminal chemoreceptors, though CCK has also been detected in the brain of sheep. Additionally, intraventricular injection of CCK in sheep is able to induce satiety (Baile et al., 1987). GLP-1 has also been implicated to play a role in intake regulation as its secretion has been associated with increase in glucose levels in plasma (through its mediation of the role of glucose transporter 2, GLUT 2 in the luminal brush border) and hence may assist in the monitoring and conveyance of information to the CNS about the energy status of the body (Faulkner and Pollock, 1991). In addition to these intestinal hormones, it has recently been shown that ghrelin (an acylated peptide located in the small intestine that is associated with releasing growth-hormone) has a major role in the regulation of intake in mammals (Hayashida et al., 2001).

The veinus drainage of absorbed nutrients yielded by a meal is funneled into the liver (although lipids are absorbed into the lymphatic system and may not directly affect liver function). The liver has extensive innervation by branches of the hepatic plexus and the vagus nerve providing a strong link to the CNS to communicate an animal's nutritional status to the brain. For instance, though infusions of glucose into the portal or jugular veins in ruminants had no effects on feed intake, Anil et al. (1993) showed that portal vein propionate infusion in sheep depressed feed intake and that denervation of the liver abolished the effect of portal vein propionate infusion and temporarily blocked transmission of impulses towards the brain in the splanchnic nerves. This demonstrates that the liver transmits its metabolic information to the CNS through the nervous system. Similar results were obtained by Langhans et al. (1985) using pyruvate, malate and lactate.

There is therefore general agreement in the literature pointing to the CNS, particularly the brain, as playing the principal role in the physiological regulation of intake, growth and energy usage in animals. The hypothalamus-pituitary-adrenal axis has been shown to be the major regions of the CNS that regulate energy balance (Boswell et al., 1998). The hypothalamus, playing the key role, works through the conveyance of hunger and satiety signals from its ventromedial section to the CNS. Several hypothalamic pathways (especially involving neuropeptides) have been implicated to exert either inhibitory or stimulatory influences on feeding (Woods et al., 1998). Among genes that have been shown to express stimulatory regulatory influences on feed intake (anabolic factors) include neuropeptide Y (**NPY**), orexins A and B (hypocretins 1 and 2), agouti-related protein (**AGRP**), melanin concentrating hormone (**MCH**), galanin, β-endophin, dynorphin, norepinephrine, growth hormone releasing hormone (**GHRH**), neuropeptide Y receptor, and galanin receptors (Boswell et al., 1998).

Regulatory pathways that inhibit feed intake (catabolic pathways) include leptin, leptin receptor, corticotrophin releasing factor (**CRF**), pro-opiomelanocortin (**POMC**), α-melanocyte stimulating hormone (α-**MSH**), GLP-1, melanocortin receptors, thyrotropin-releasing hormone, and cocaine and amphetamine regulated transcript (**CART**) (Houseknecht et al., 1998; Woods et al., 1998; Baile and Della-Fera, 2001). Other hormones with significant direct or indirect effects on intake regulation and energy partitioning in livestock include growth hormone (**GH**), growth hormone receptor (**GHR**) insulin, insulin-like growth factors (**IGFs**), IGF binding proteins (**IGFBP**), thyroid hormones, adrenergic receptors, as well as androgens and estrogens (Breier, 1995). Most of these (with the exception of the latter three groups) are regulated through the somatotropic axis, which functions as a mediator for adjusting and controlling hormonal responses to growth and affect and interact with other hypothalamic pathways to regulate energy balance in animals. Yet other genes that have been implicated in feed intake regulation through appetite regulation include carboxypeptidase E, the PPARg2 gene, uncoupling proteins, apolipoproteins, and tumor necrosis factor (**TNF**) (Roberts and Greenberg, 1996). The regulatory effects of all these effector molecular pathways are integrated with other pathways that have an influence overall energy balance and tissue metabolism in the mammalian system. This complexity has rendered the mechanisms involved in the regulation of feed intake and energy balance a highly redundant system.

2.5.2 Leptin and the regulation of feed intake and energy partitioning

The relative contribution of each of the candidate genes listed above to the regulation of feed intake and energetic efficiency are presently unknown. Perhaps the most extensively studied gene expressed by hypothalamic neurons in relation to the regulation of feeding is leptin, a 146-amino acid cytokine-like peptide product of the obese gene that is expressed primarily in adipose tissues (Zhang et al., 1994). There is literature evidence pointing to several possible complex interactions between leptin and other substrates in the body, especially hormones, creating a complex network of feedback loops between leptin and the hypothalamic genes encoding neuropeptides and metabolic hormones. For instance, there is some evidence that both growth hormone and thyroid hormone affect leptin synthesis and/or secretion. It has also been shown that changes in leptin mRNA and serum levels are a result of an independent effect of thyroid hormones on adipose stores (Syed et al., 1999; Anderson, 2000). A recent study showed that growth hormone treatment in rats reduced leptin mRNA levels in certain fat tissues, indicating that GH directly interacts with fat tissues to lower leptin gene expression (Woods et al., 1998 and Isozaki et al., 1999). It is also known that the inhibitory actions of leptin are generally opposed by those of glucocorticoids, depending on species, and these two hormones exert reciprocal influences on each other's secretion. Additionally, leptin and insulin are known to process feeding-related signals from the GIT such as those originating from the peptide CCK (Forbes, 2000). A relation has been established between plasma leptin and insulin, where insulin stimulates leptin gene expression in adipose tissues whereas leptin regulates glucose metabolism and insulin action (Houseknecht et al., 1998). Similar relationships have been established between leptin and other hormones such as adrenergic receptors (Bachman et al., 2002).

The negative effects of leptin on feed intake are achieved in part by stimulating gene expression of catabolic neuropeptides and inhibiting that of anabolic neuropeptides (Woods et al., 1998). Certain classes of catabolic and anabolic neurons have been reported to both interact with and/or account for leptin sensitivity in the central nervous system. Significant among these include those activated by leptin as represented by CRF, POMC, and CART. There are also those peptides whose effects are negatively affected by leptin as represented by NPY, AGRP, and MCH (Baile and Della-Fera, 2001; Rahmouni and Haynes, 2001).

Several studies have implicated leptin in metabolic regulation through its action on both the hypothalamic-pituitary-adrenal axis activity and on reproductive function. A product of the *ob/ob* (obesity) gene, leptin is synthesized by white adipocytes. After synthesis and secretion, leptin crosses the blood-brain barrier through a saturable unidirectional specific transport mechanism, where it binds to cells expressing the leptin receptor to inform the CNS of the energy status of the body (Houseknecht et al., 1998). Leptin receptors are single transmembrane proteins belonging to the cytokine superfamily. They have been shown to be highly expressed in several hypothalamic nuclei including those of the arcuate, ventromedial, paraventricular, and dorsomedial hypothalamus (Tartaglia et al., 1995).

Leptin appears to have a number of functions, mostly related to body energy homeostasis. It functions as an efferent regulator through both central and peripheral pathways to affect feeding behaviour and modulate food intake (by appetite reduction), lipid and glucose metabolism and energy expenditure (by thermogenesis) through sympathetic stimulation to brown adipose tissues (Baile et al., 2000). Other physiological roles played by leptin include regulation of reproduction, sexual maturation, the overall hypothalamic-pituitary-adrenal system, thyroid and growth hormone axis, haematopoiesis, cardiovascular and immune system functions, and bone remodeling (Amling et al., 2000; Baile et al., 2000). Obesity is associated with increased leptin synthesis and secretion whereas fasting and weight loss are associated with decreased leptin synthesis and secretion (Houseknecht et al., 1998).

Leptin binding to its receptor in the hypothalamus is essential to the regulation of food intake and energy expenditure in animals. A remarkable aspect of the catabolic response to leptin administration is that the weight loss appears to be due entirely to loss of body fat. Some studies with normal, lean, animals show that continuous leptin administration can virtually eliminate detectable body adipose stores because of a relative increase in the rate of metabolism coupled with reduced energy intake (Woods et al., 1998). Evidence also shows that response to the inhibitory feedback effects of leptin is more sensitive in leaner animals, and sensitivity is greatly reduced in animals with large fat stores, even though circulating concentrations of leptin in the latter group are high (Houseknecht et al., 1998). This is the basis for the not-yet understood phenomenon of leptin-resistance in certain obese animals that is recently attracting considerable research attention. It has been suggested that some of the leptin receptor forms may be involved in leptin resistance (Houseknecht et al., 1998).

The major hypothalamic site of transduction of afferent input from circulating leptin into a neuronal response is the arcuate nucleus (Houseknecht et al., 1998). This is evident in the fact that there is a concomitant decrease in food intake induced by local injection of leptin into this region of the hypothalamus (Rahmouni and Haynes, 2001), compared to effects observed in other routes of administration. According to these authors, there is also literature evidence indicating that central neural leptin administration does not affect intake after the arcuate nucleus has been destroyed. Leptin alters the transcription of several adipose-specific genes involved in lipogenesis, lipolysis, and energy metabolism, and seems to trigger apoptosis in white adipose tissue (Qian et al., 1998). Several studies in ruminant livestock have shown that the roles played by leptin in feed intake and energy regulation in humans and rodents are similar to those in ruminant livestock.

In well-fed ruminant animals, central administration of leptin reduced food intake (Morrison et al., 2001) and energy intake level was positively related to adipose tissue leptin mRNA (Amstalden et al., 2000). Studies with cattle (Chilliard et al., 1998; Delavaud et al., 1999; Amstalden et al., 2000; Luna-Pinto and Cronjé, 2000; Wegner et al., 2001; Ren et al., 2002; and Delavaud et al., 2002) and sheep (Boquier et al., 1998; Kumar et al., 1998) indicated that the amount of feed consumed and body fat are closely related with plasma leptin concentration. For instance, Amstalden et al. (2000) studied the responsiveness of leptin gene expression, circulating leptin, and other hormones to short-term fasting in prepubertal heifers. Their results showed that leptin gene expression and circulating concentrations were significantly lower in fasted heifers compared to control heifers.

Plasma leptin concentrations of restricted-fed heifers were also observed to be lower than control groups in the study by Luna-Pinto and Cronjé (2000). These authors also observed more variability in plasma leptin levels in the control heifers compared to the restricted group. In this same study, FCR was higher over time for control groups than for restricted groups, confirming the observed relationship between weight gain and plasma leptin levels. There seems to be significant genetic variation in leptin intervention in livestock species. In beef cattle, Wegner et al. (2001) reported that significant differences in plasma leptin concentration for crossbred cattle of varying Wagyu cattle (WC) genetics were in the order 3.85, 7.50, 8.78 ng/mL for 0%, 50%, and 75% WC genetics, respectively. Generally, accretion type cattle (beef cattle) accumulated less body fat and show a lower plasma leptin concentration than secretion type cattle (dairy cattle). However, the plasma concentration of leptin in lean dairy cattle is generally lower than that in fat beef cattle.

Recent studies by Ren et al. (2002) using Holstein and Charolais bulls showed that the amount of leptin mRNA in subcutaneous and perirenal adipose tissues was higher in

Holstein bulls than in Charolais bulls. It appears that, depending on the amount of adipose tissue, the ability of the body to regulate leptin secretion is altered. The higher the ability to transform nutrients into fat, the lower the regulation is via leptin. This evidence therefore not only points towards breed differences in leptin levels, but also shows that leptin expression in the body occurs in a manner proportional to the amount of body fat (Baile et al., 2000). Studies conducted using obese, diabetic, and sterile mice that exhibited reduced activity, metabolism and body temperature showed that daily leptin administration resulted in decreased food intake, body weight and body fat, normalized serum glucose and insulin levels, restored fertility and increased metabolic rate, as well as body temperature and activity levels compared to those of lean litter mates (Pelleymounter et al., 1995).

Thus, leptin functions as a lipostatic signal regulating body weight, feed intake, energy expenditure (Houseknecht et al., 1998, Woods et al., 1998), reproduction (Cunningham et al., 1999, Garcia et al., 2002) and immune system functions (Lord et al., 1998). Circulating leptin and tissue mRNA levels are correlated with body weight, food intake, nutritional status and adipose tissue mass in humans and animals (Larsson et al., 1998, Amstalden et al., 2000, Delavaud et al., 2002). In cattle, circulating leptin levels are correlated with the regional distribution of body fat (Yamada et al., 2003) and carcass merit and composition (Minton et al., 1998, Geary et al., 2003) though other studies using mainly Japanese Black cattle (Kawakita et al., 2001, Tokuda and Yano, 2001) did not find any relationships of serum leptin with body fat.

Although data exist in ruminants pertaining to the effect of short-term fasting, feed restriction and leptin administration on body weight and intake (Amstalden et al., 2000, Morrison et al., 2001, Ren et al., 2002) little is known about the relationship of endocrine leptin with the growth and voluntary feed intake of feedlot cattle. Most of the studies cited above either used relatively fewer animals under fasting or underfeeding treatments. Garcia et al. (2002) showed a correlation between body weight and serum leptin concentration in heifers to be 0.85. Ehrhardt et al. (2000) observed that cattle on a higher plane of nutrition grew faster, had higher body fat, which was associated with elevated (46%) plasma leptin concentration. Kawakita et al. (2001) however showed no relation of plasma leptin with feed intake. Thus, in the present work, attempts would be made to evaluate the relationships of endocrine leptin and polymorphisms in the leptin gene with measures of performance, feed efficiency and carcass merit in beef cattle.

2.5.3 Effect of digestibility and metabolizability

The processes of digestion, absorption, retention and excretion of nutrients in mammals, especially ruminants, are made up of several complex processes that may be influenced by a wide range of factors. Techniques employed in nutritional energetics of ruminants have classically been concerned with the partitioning of dietary energy into faecal, urinary, methane, heat, and recovered or product energy. How well an animal can digest and assimilate nutrients for productive purposes depends upon the bioavailability of the nutrients in the diet, as well as absorption capacity of the digestive tract, metabolism and retention (Church and Pond, 1988). However, it is the bioavailability of the nutrient in the diet that would ultimately determine how much of the ingested feed would be voided as faeces. Three main factors affect the amount of nutrient losses in faeces, namely amount consumed, efficiency of conversion to digestible energy (between animal variations in digestibility) and amount of endogenous secretions.

The primary opportunity for reducing the amount of nutrient excreted by animals is to reduce dietary intake but at the same time optimize the efficiency of utilization of ingested nutrients (Paul et al., 1998). Literature evidence on dairy cattle indicates that only 21-24% of ingested nitrogen (on a whole herd basis) is recovered in milk and meat products (Wright et al., 1998). The proportion of intake nitrogen not recovered in products is not due only to variations in digestibility as it is known that protein digestibility is usually very high in cattle (Church and Pond, 1988). Instead, a larger proportion of the feed nitrogen is excreted in the urine than in the faeces and urinary nitrogen concentration is strongly affected by feeding. All the absorbed amino acid nitrogen that is not needed for milk or meat production will end up in urine.

The implication of this is that even animals that are efficient in the conversion of dietary nitrogen into metabolizable nitrogen may still be inefficient depending on how much of the absorbed proportion ends up as net energy for maintenance or production. There is limited literature evidence regarding variations between animals in retention of dietary nutrients for growth and production. A study by Kauffman and St-Pierre (1999) indicated no significant differences between Holstein and Jersey dairy cattle breeds in the amount of milk urea nitrogen and apparent DM digestibility of feed. The same report however, indicated that for animals of the same breed, higher producing animals have significantly higher milk urea nitrogen, higher nitrogen intake, higher faecal nitrogen excretion, higher milk nitrogen, higher nitrogen retention, and higher apparent nitrogen digestibility.

Zinn (1994) reported significant effects of a 20% variation in daily feed intake on faecal excretion of organic matter, starch, ADF, and nitrogen in Holstein steers. Recent estimates by Okine et al. (2001) indicated that low net feed intake growing beef steers had significantly lower manure production per unit gain, as well as significantly lower nitrogen, phosphorous and potassium excretion per 550 kg of gain. These differences may indicate genetic variations in the amount of dietary nutrients excreted or retained in the body for productive function. The goal of efficient and productive feeding of animals, within economic and environmental constraints, is to provide essential available nutrients for maintenance and production with minimal excesses and losses. Therefore, any small differences detected in the digestibility or availability of nutrients between animals could be exploited in selection programs to increase the potential for animal use and reduce the amount of losses in the overall production system (Luiting, 1999).

Two possible sources of variation in feed efficiency between animals, in addition to digestibility, may originate from energy losses in the form of heat and gaseous products of fermentation between the conversions of DE to NE. The NE of food is the difference between ME and heat increment due to feeding and fermentation (in ruminants). Heat increment (or specific nutrient dynamic effect) is the heat production associated with nutrient digestion and metabolism over and above that produced prior to ingestion (Church and Pond, 1988). In cattle, heat increment has been estimated to be about 3% of GE for a complete diet at half maintenance and may increase to over 20% at higher levels of maintenance. This has important implications for beef production, especially in the breeding herd. The lower overall efficiency of beef production, compared to dairy systems, may be due to the fact that maintenance energy is a greater proportion of total dietary energy in beef cows (Blaxter, and Wainman, 1966). Several techniques are available for measuring heat production in animals. A direct calorimetry technique involves enclosing an animal in a well-insulated chamber and measuring heat loss by means of thermocouples, circulation of water in pipes in the chamber or by electrical means using gradient layer calorimetry (Church and Pond, 1988). Indirect measurements of heat production are based on the determination of oxygen consumption, carbon dioxide and methane production (Delfino et al., 1988; Young et al., 1984). Several techniques have been developed for estimating heat production from these measurements (McLean and Tobin, 1990).

There is evidence indicating considerable genetic variations in the amount of energy lost as heat in animals. Blaxter and Wainman (1966) indicated that average values of heat increment vary between Ayrshire steers (100 kcal/BW$^{0.73}$) and Angus steers (81 kcal/BW$^{0.73}$); similar differences were also observed between breeds of sheep and dairy cows. A recent estimate by Basarab et al. (2003) indicated that heat production by low RFI beef steers (122.8 kcal/kg$^{0.75}$) was 11.2% lower than medium RFI (138.4 kcal/kg$^{0.75}$) and 17.0% lower than high RFI steers (147.9 kcal/kg$^{0.75}$). These represent marked variations between animals in heat production that may greatly account for genetic variations in feed efficiency, and which can be exploited to increase production system efficiency.

Methane and other gaseous products of enteric fermentation represent substantial losses in dietary energy from cattle (Luiting et al., 1994). Methanogenic microbes such as *Methanobrevibacterium ruminantium*, *Methanobacterium formicicum*, and *Methanomicrobium mobile* produce copious quantities of methane in the rumen (Church and Pond, 1988). Methane produced during ruminal fermentation is belched by the animal, and accounts for the majority of the emissions of greenhouse gases from ruminants. A number of factors affect methane production in ruminants, including the physical and chemical nature of the diet, feeding level, feeding schedule, addition of feed additives, the activity and health of the animal, as well as genetic differences between animals (Gibbs and Leng, 1993). There is a complex relationship between the quantity of feed consumed and the percentage yield of methane. Methane production, as a fraction of gross energy intake, generally decreases as daily intake increases for the same diet, but the actual quantity of methane may increase due to greater amount of digesta in the rumen.

It is generally estimated that conversion rate of dietary energy to methane is about 5.5-6.5% (range of 2-12%) of GE intake for most well fed cattle in temperate agricultural systems (Gibbs and Leng, 1993). Several reports in the literature have indicated the existence of genetic variation between individual animals in methanogenesis. Jarosz and Johnson (1999) reported significant variations in methane production between Hereford steers and Hereford crossbred steers fed fibrous by-product feeds. Okine et al. (2001) estimated methane emissions from high, medium and low RFI steers. Their results indicated no significant differences in methane emission as a percent of GE among the animals. However, daily methane production (in g/day or Mcal/day) was significantly higher in higher RFI steers than for low RFI steers. Additionally, yearly methane emissions from low RFI steers were estimated to be 21% lower than for high RFI steers. These are evidence of marked genetic differences in efficiency of feed utilization and have significant implications for the overall production system efficiency.

2.5.4 *Effect of absorption and transport of dietary nutrients*

Absorption of dietary nutrients takes place in the upper section of the small intestine, including the duodenum and the jejunum, and to a lesser extent in the ileum. In cattle and other ruminants however, VFAs produced from ruminal fermentation is mainly absorbed across the walls of the stomach epithelium where they are carried by ruminal veins to the portal vein, and hence through the liver. Though post-gastric fermentation does occur in cattle, and certain vitamins, electrolytes, water and other substances may be absorbed, absorption in the large intestine is not considered under this review. The passage of individual nutrients into the blood stream or lymph from the lumen of the GIT may occur by passive diffusion, by active transport, or by pinocytosis (that is, the engulfment of large particles or ions in a manner similar to endocytosis) (Matthews, 2000; Russell and Gahr, 2000). Passage by diffusion or by active transport involves the penetration of the microvillus and of the plasma membrane through the cell interior, possible metabolism within the cell, extrusion from lateral and basal aspects of the cell, passage through the basement membrane or lymphatic epithelium into blood or lymph (Mathews, 2000). The exact mechanisms and systems of absorption and transport of proteins, carbohydrates and lipids are elaborated below.

Absorption of amino acids and peptides from the intestinal lumen takes place in the duodenum, jejunum, and ileum by active transport through the brush border membrane (**BBM**). The transport systems that mediate the passage of amino acids and peptides across plasma membranes of non-polarized cells and the apical and basolateral membranes of polarized epithelial cells also mediate the passage of substrates across the membranes of cell organelles such as lysosomes, mitochondria, and the nucleus (Mathews, 2000). Generally, amino acid and peptide transport systems with relatively low affinities (ability to recognize and bind substrate molecules) for substrates have large capacities for transport, but those that demonstrate high affinities have low transport capacities (Ganapathy et al., 1994). The transport process requires energy that is provided by the hydrolysis of ATP (from cellular respiration) or by trans-membrane potential differences.

Absorption of VFAs, "waste products of rumen microbial fermentation", takes place through the squamous epithelial lining of the stomach down a concentration gradient (Church and Pond, 1988). The mechanisms involved in VFA absorption have been reviewed comprehensively by Gabel (1995) and Rechkemmer et al. (1995). Previously, VFA absorption was thought to be mainly by passive diffusion of undissociated acids through the rumen epithelium (Maynard and Loosli, 1969). In this case the rate of absorption is influenced by the pH of the lumen of the gut and the chain length of the acids, but level of influence is very highly dependent on differences in absorption rates observed at different concentrations of VFAs. It has been reported that, though passive diffusion of undissociated acids occurs, active anion uptake coupled with exchange of intracellular bicarbonate with luminal anion is the predominant mechanism of VFA absorption (Rechkemmer et al., 1995). There is also evidence that anionic uptake is mediated by luminal $CL^-/Na^+/H^+$ exchangers and a Na^+/K^+ ATPase, though the stoichiometric relationships of these have not been determined (Russell and [illegible]ahr, 2000).

With respect to carbohydrates that reach the small intestine, the duodenum and the jejunum have the greatest capacity to absorb monosaccharides, especially glucose and galactose (Church and Pond, 1988). According to these authors, active transport has been established for glucose and galactose in the small intestine, but may not exist for other sugars. It has also been reported in the literature that certain glucose transporters exist that mediate the uptake of fructose (Burant et al., 1992). Two major routes (transcellular and paracellular routes) have been proposed and studied extensively as being the mechanisms through which carbohydrate absorption and transport occurs in the small intestines (Harmon and McLeod, 2001). The paracellular route of transport has been shown to be dependent on the transcellular route (Wright, 1993). This transport mechanism has been shown to be accomplished by glucose transporter 2 (**GLUT2**). The system is stimulated by the accumulation of solutes of active transport (Na^+, glucose, amino acids, free fatty acids, etc.) around enterocytes (Ballard et al., 1995). The major transcellular route for glucose and galactose transport in the small intestine is the Na^+-dependent co-transport system (**SGLT1**, Wright, 1993).

It has been shown that apical membrane events of the SGLT1 transport system follow an ordered mechanism and the affinity of SGLT1 for Na^+ is dependent on the electrochemical potential of the membrane (Ferraris et al., 1990). Na^+ is the primary solute without which the transport of glucose may be greatly impaired. Another type of transepithelial transport system for sugars (glucose transporter 5, **GLUT5**) has been shown to exist. GLUT5 catalyses insulin-independent facilitated diffusion of monosaccharides across the apical membrane of enterocytes (Olson and Pessin, 1996). Evidence from some of the above reviews point out that GLUT5 is the major transporter for fructose uptake, which occurs on the apical membrane of enterocytes from approximately mid-villus to the tip. Fructose uptake in this manner has been shown to be uninhibited by the presence of glucose.

Carbohydrate absorption and transport from the small intestine of cattle, and other ruminants, has been shown to be less effective compared to direct absorption from the gut of monogastric animals (Okine et al., 1994). According to Huntington (1997) limiting luminal and membrane capacity is the major factor responsible for this. Additionally, it has been reported that there is ceasation of expression of SGLT1 in ruminants post weaning, and this may also be a major factor contributing to less efficient glucose transport in cattle and other ruminants. Also, studies on lactating and feedlot cattle consuming high levels of cereal grain diets (as are found in North America) have indicated relatively high abundance of SGLT1 mRNA in the rumen, omasum and cecum, as well as in the small intestine of cattle (Zhao et al., 1998).

With regards to lipid absorption and transport, the bile salt/lysolecithin micelle associate with the brush border of the intestinal epithelium and facilitates the transport of the hydrophobic fatty acids across the BBM. Fatty acids are re-esterified in the small intestine to glycerol-3-phosphate (glycerol-3-phosphate is made through glycolysis from blood glucose) to form triacylglycerols. Along with apolipoproteins (B48, AI and AIV), cholesterol and phospholipids, the triacylglycerols are packaged into lipoprotein particles (analogous to chylomicrons in monogastrics) that are secreted from the cells and enter the lacteals to be carried into the lymph and reach the peripheral circulation. These apolipoproteins are usually known as very low-density lipoproteins (**VLDLs**) (Hussain et al., 1996). During transport and metabolism, VLDLs secreted from the intestine or liver acquire apoprotein CII (**apo-CII**) from newly secreted high-density lipoproteins (**HDLs**). The triacylglycerols in the VLDLs are hydrolyzed by lipoprotein lipase (**LPL**) in peripheral tissues, which is activated by apo-CII and allows fatty acid uptake by tissues. The remaining particles, known as intermediate-density lipoproteins (**IDLs**), are either cleared by the liver or undergo further triacylglycerol hydrolysis to produce low-density lipoproteins (**LDLs**) (Braun and Severson, 1992). The excess surface components, namely phospholipids, apoproteins C and A, free cholesterol, are transferred to HDLs. HDLs may take up excess cholesterol from peripheral tissues and convert it to cholesterol esters by the action of lecithin cholesterol acyltransferase (**LCAT**). This allows lysolecithin to be released into blood plasma, and cholesterol esters enter the core of HDLs. HDLs can deliver cholesterol to tissues or return it to the liver for conversion to bile salts (Drackley, 2000).

Thus, the mechanisms of absorption and metabolism of carbohydrates and related substances, amino acids and peptides as well as lipids have been well characterized and documented in ruminants. However, there seems to be little or no reports in the literature characterizing the magnitude of differences between individual animals or different breeds of livestock. In dairy cattle, Russell and Schmidt (1984) reported that the variation in the absorption of intestinally infused glucose ranges between 10-100%. In beef cattle, Harmon (1992) indicated that variations between individual beef steers in intestinal starch disappearance might be as high as 10-93%. Considering the several mechanisms and processes involved in absorption, transport and metabolism of peptides, amino acids, lipids, and carbohydrates, there is the possibility of variations in efficiency of nutrient transport between individual animals. Any new evidence regarding actual differences between animals in absorption and transport efficiency would be highly useful in the characterization of metabolically efficient animals.

2.5.5 *Physical activity and behaviour*

The most recent data available in the literature on the physiology of eating as related to the energy expenditure of livestock species dates back to the 1970s. Direct and indirect calorimetry techniques (Blaxter, 1989; Wainan and Blaxter, 1958) have been used to estimate the energy requirements of animals reared indoors. Though attempts to obtain similar estimates for free ranging animals are much more complicated, there are reports in the literature (Blaxter, 1989) pointing to the extent of total energy requirements in animals kept out doors. According to Blaxter (1989), the maintenance requirements of sheep and cattle at pasture are 11% and 15% higher than those indoors, possibly due to increased costs of body movements at pasture, the effects of the outdoor environment, or errors due to inconsistencies in measurements outdoors.

It was suggested that the potential for increased energy expenditure at pasture might be due to increased overall costs associated with grazing, especially the costs of walking to and harvesting the herbage, which depend on the availability of the pasture and environmental stressors (Osuji, 1973). According to Graham (1965), the energy expended in muscular work by sheep at pasture could be up to six times that of housed sheep due to standing, walking, eating, the energy cost of rumination, and the secretory activities associated with feeding (ARC, 1965). The energy cost of eating has been said to constitute an appreciable part of the extra maintenance requirement of a grazing animal. The muscular activities of prehension and mastication plus the secretory activities associated with feeding are essential components of this complex. Thus the study of the marked changes associated with eating, especially their contributions to the overall heat production of an animal may lead to a better understanding of the physiology of forage utilization, particularly with regards to energy requirements and the effects of the physical form of the diet on the productivity of ruminants.

The maintenance energy requirement of an animal is usually defined to include normal activity. However, many factors such as housing, space allowance, feeding, and temperature in addition to the individual variation that may occur between individual animals may affect physical activity. Different techniques exist to quantify activity (standing versus lying, measurement of movement or force) leading to different estimations of the energy expenditure in the active and non-active states. Though in general, the energy an animal spends in an enclosure in minor movements is very small and may not reflect their activity under normal conditions, the energy cost of standing versus lying may be very significant, especially in animals at pasture (Osuji, 1974).

This is especially important when the availability of pasture is limited. It has been shown in both sheep and cattle by several investigators (as reported by Osuji, 1974) that there is a linear increase in grazing time as pasture availability decreases. Thus differences in energy expenditure at pasture and in an enclosed chamber depend on the cost of the activity and the time spent in pursuing them (Osuji, 1974). The energy spent in walking has been determined for several livestock species. In sheep, horizontal locomotion has been shown to increase with speed and averages 0.59 cal/kg m whilst vertical locomotion is about 10 times higher (6.45 cal/kg m). Though ARC (1965) considers the energy cost of eating to be negligible and therefore considers the heat increment of feeding in ruminants to be mainly accounted for by the heat of fermentation and the energy cost of metabolizing VFAs, this has been shown to be unlikely (Christopherson, 1971; Christopherson and Webster, 1972). These reports show that grazing and feed intake as a whole is probably energetically more expensive and the cost of grazing may explain the differences between published accounts of the maintenance energy requirements of the grazing animal.

The energy cost of eating varies directly with the time spent in eating (r = 0.86) and it is estimated that the energy cost of eating for housed animals accounts for 2-3% of their daily energy expenditure (Osuji, 1974). For free ranging animals, calorimetric estimates show that grazing requires 10-15% more energy for maintenance than in a housed animal, and estimates as high as 40% have been proposed (Blaxter, 1989; Osuji, 1974). There is therefore, a strong suggestion that energy cost of eating makes a significant contribution to the energy requirements of animals, especially those at pasture. The work done in digestion involved in the handling of bulky fresh grass might account for an appreciably high fraction of the total heat increment of feeding observed in ruminants (Osuji, 1974). Additionally, the fluid and electrolyte changes and their flow into the gut (Christopherson and Webster, 1972) as well as cardiovascular changes associated with eating in terms of arterial carbon dioxide and other gaseous tensions may account for significant fractions of the energetics of feeding.

The specific relationships of measures of activity, especially feeding behaviour, with many economically important traits in farm animals such as performance and feed efficiency are not well-known. Keys et al. (1978) showed that in yearling Holstein heifers, feeding duration is only weakly related to feed intake. However, most previous studies documenting the patterns of feeding behaviour were based on observations from groups of animals (pen mates), which may be lower in accuracy and sensitivity. Using automatic feed intake and behaviour monitoring equipment, (based on radio-frequency technology), Schwartzkopf-Genswein et al. (2002) reported a moderate relationship between feeding duration and feed intake. Robinson and Oddy (2004) reported heritability estimates for measures of feeding behaviour in feedlot cattle ranging from 0.36 to 0.51. The same authors showed that measures of feeding behaviour were genetically and phenotypically related to measures of performance, efficiency, and carcass merit. Genetic variation in feeding behaviour have also been reported in sheep (Cammack et al., 2005) with heritability estimates ranging from 0.29 to 0.36. The same authors also showed considerable genetic and phenotypic relationships between the measures of feeding behaviour with measures of performance and feed efficiency.

2.5.6 *Tissue metabolism and energy partitioning into fat and protein.*

The study of cellular energetics, and therefore maintenance energy requirements, is the first step towards the understanding of the variations in overall requirements of animals and the factors that influence it (Jessop, 2000). Maintenance requirements include BMR, energy costs of certain muscular activities (locomotory costs and costs associated with eating and processing of food within the digestive tract), and energy costs for operation of the immune system and fighting infection as well as in thermoregulation during shivering and non-shivering thermogenesis (Jessop, 2000). Several techniques have been employed over the years to determine energy metabolism and expenditures in farm animals and these techniques are constantly evolving. Balance studies using markers, *in vitro* fermentations and laboratory analysis of the chemical characteristics of feedstuffs have allowed the characterization of energy transformations in animals from prehension to excretion (Noziere and Michalet-Doreau, 2000). Direct and indirect calorimetry techniques have enabled the determination of total energy expenditures through the measurement of respiratory and other gases (Blaxter, 1989). The use of comparative slaughter techniques have been employed to measure retained energy in various tissues. Indirect respiratory calorimetry and comparative slaughter techniques continue to be important energetics research tools (Reynolds, 2002).

Techniques utilizing blood flow, thermal dilution, and gas analyses to quantify and separate heat generated from the GI tract into aerobic and anaerobic origins have been successfully applied to ruminants. Measurement of oxygen arterial-venous concentrations has allowed the direct quantitation of energy expenditure of various tissues and organs (Reynolds, 1995). Additionally, a number of *in vitro* techniques utilizing tissue or cell preparations have enabled more detailed energy measurements. Assessment of tissue energy metabolism from blood flow and substrate flux across the PDV, liver, gravid uterus, fetus, mammary gland, and hind limb have contributed substantially to present understanding of tissue energy expenditures and sources of their variation (Seal and Parker, 2000). Generally, the BMR component of maintenance energy requirements may be subdivided into those required for service functions (energetic cost of organ systems, namely kidney work, heart work, respiration, nervous functions, and liver functions) and for individual cell maintenance (specifically ion transport, protein turnover and lipid turnover) (Baldwin, 1995). Most visceral tissues are characterized by high rates of metabolism, and these high rates arise primarily through their many support and service functions (Freetly and Ferrell, 1997).

Early published data on the energy expenditures of various tissues and organs of the body indicated considerable variability depending on the technique used. Data published by Smith (1970) indicated higher energy expenditures for tissues such as those of the GIT, muscle, liver, nervous tissue, and the heart; relatively lower expenditures by adipose tissues, skin, kidneys, and other skeletal tissues. These relative published estimates were obtained partly in concert with the relative rates of tissue turnover and transport activities and may be significantly variable under real situations. Though organs such as the heart, the brain, the lungs and their associated tissues may have significant tolls on the overall energy expenditure and metabolism in the body, tissues of the splanchnic bed have been given much recent research attention probably due to the fact that these tissues are established at the crossroads of energy transformations and play major roles in energy metabolism in the body.

The total splanchnic tissues include the kidneys, the portal drained viscera (PDV; gastrointestinal tract, pancreas, spleen, and associated omental and mesenteric fat), and the liver. These, together with the associated connective tissues and blood vessels, have been shown to form approximately 15-20% of total body mass in ruminants (Seal and Parker, 2000). They collectively play an important role in the metabolism of dietary energy and moderate the supply of nutrients to peripheral tissues for maintenance and a number of productive processes. They therefore account for substantial portions of body oxygen consumption and carbon dioxide production (total energy expenditure) in the body, mainly due to the rate of protein turnover in these tissues. Indeed, Webster (1980) indicated that there is a strong linear relationship between protein synthesis and heat production and that marked differences in metabolic rate could almost entirely be explained by differences in protein synthesis.

Not surprisingly, the greatest proportion of the protein synthesis and associated heat production takes place in visceral tissues such as the gastrointestinal tract and the liver, which are not normally associated directly with growth and meat production. Specifically, splanchnic tissues have been shown to account for between 36 and 54% of body oxygen consumption in growing and lactating cattle (Reynolds, 1995), but this varies with both dry matter and metabolizable energy intake. Additionally, metabolism of the splanchnic tissues account for a substantial proportion of body heat production. For instance, literature evidence from growing heifers indicate that total splanchnic tissues account for about 44% and 72% of the incremental rise in body oxygen consumption for two different diets, respectively. It has been reported that within the limits of variability due to diet composition and productive state, considerable amounts of variability exists between ruminants in splanchnic oxygen consumption (Reynolds, 2002).

In ruminant livestock, especially cattle, energy expenditure by the kidneys have been shown to represent a true maintenance energy cost (Reynolds et al., 1991). Though the kidneys generally receive more blood per unit weight than any of the tissues of the splanchnic bed in ruminants, the high rate of blood flow has been shown to be related to their inherent role in the excretion of waste products and maintenance of fluid balance

(Reynolds, 2002). Therefore, their fractional extraction of oxygen is much lower than for tissues such as the liver. For instance, Reynolds et al. (1991) report that the kidneys account for about 6% of total body oxygen consumption irrespective of diet composition or level of intake.

Anatomically, the liver is positioned at the crossroads of the body and therefore controls the quantity and characteristics of nutrients available to the peripheral tissues from digestive tract absorption. It constitutes less than 2% of the overall body mass of adult ruminants, but receives approximately 25% of the total cardiac output, depending on nutrition and other regulatory factors. Some of the major roles of the liver include production and removal of key hormones, hepatic detoxification to prevent hyperammonnaemia, regulation of systemic plasma amino acid concentration, in addition to the general functions of intensive nutrient assimilation and waste management involving both the liver and the PDV (McBride and Kelly (1990).The energy expenditure of the liver responds to general services as well as to nutrient requirement and dry matter intake (Reynolds, 1995).

The functions of the liver in the synthesis of numerous compounds and its roles in waste management and detoxification dominate the metabolic activity of the liver. Additionally, cell maintenance and nutrient transport require substantial levels of ion transport, which is a highly energetic process (Reynolds, 2002). These numerous service functions lead to substantial energy expenditure in the form of oxidizable substrates, mainly acetate, glucose, beta-hydroxybutyrate and long chain fatty acids that account for much of the body CO_2 production (Annison and Bryden, 1999). However many non-essential amino acids as well as excess available amino acids may be subjected to catabolism and oxidation and provide ATP through specialized inter-organ shuttles (Reynolds, 2002).

Literature evidence shows that in dairy cattle, the liver receives more than 40% of total blood flow and account for 20 to 25% of total body energy expenditure (Reynolds, 1995; Annison and Bryden, 1999). It has been shown that nearly all the butyrate, propionate and VFAs absorbed are removed and utilized by the liver. Additionally a significant proportion of glucose and amino acids reaching the liver may be removed and used by the liver. The level of liver amino acid utilization may be evident from that fact that the high rate of metabolism of the liver may be associated with a high rate of protein turnover, especially for the synthesis and breakdown of constitutive and export proteins. Indeed, literature reports indicate that values as high as 20% of total dietary amino acid is extracted and metabolized by the liver (Reynolds, 2002).

The portal drained viscera (PDV) play similar roles as the liver in terms of nutrient assimilation and waste management. Energy expenditures of the PDV include service functions in terms of diet digestion, gastric motility, synthetic and secretory processes for several digestive enzymes and hormones by the spleen and pancreas, nutrient absorption and metabolism, maintenance of gut epithelial structure, and immune system functions. Additionally, the roles of cell maintenance and nutrient transfer require high levels of ion transport activity, which is highly energetic in nature. Provision of energy for all the numerous roles of the PDV may be in the form of extracted VFAs, glucose, amino acids and several fatty acids (Reynolds, 2002).

Of the approximately 35 to 60% total body oxygen consumption attributable to the PDV, the GIT alone accounts for approximately 20% (Cant et al., 1996). Tissues of the GIT form about 10-13% of total body mass and this gives a considerable level of metabolic activity, as detailed above. According to McBride and Kelly (1990) of the amount of energy consumed by the GIT, 30-60% is for Na^+, K^+-ATPase-linked ion transport activity whilst protein synthesis and protein degradation account for 20-23% and 4%, respectively. Several factors have been shown to influence GIT energy expenditure including meal consumption (Christopherson and Brockman, 1989), energy intake level (Goetsch, 1998), physiological state, increased intestinal transport capacity leading to synchronous up regulation of all intestinal transporters, and the indirect effects of hepatic ureagenesis (Reynolds, 2002).

With such considerable variations in visceral energy expenditure and the different energy substrates involved in paying for the energy cost of several of the service functions provided by tissues of the splanchnic bed, it is probable that considerable genetic variation exists in the total heat increment of feeding, digestion and energy metabolism. According to Jessop (2000) breed variability in the maintenance needs of cattle indicate the existence of considerable variation in metabolic efficiency in terms of differences in the energetics of many metabolically active tissues. The extent of such metabolic variations may be required in the assessment of the molecular and genetic basis of differences in energy use between animals differing in genotype.

Much of the available work in the area of the energetics of ion transport has been published by the research group of Milligan and McBride. Most of these estimates come from *in vitro* studies of the rate of oxygen consumption by the Na^+, K^+-ATPase system by making use of a specific inhibitor of the sodium pump, ouabain. The Na^+, K^+-ATPase enzyme system is present in the membranes of cells and serves to main the ionic gradient of Na^+ between the intra- and extracellular space at the expense of ATP (Milligan and McBride, 1985). The activity of this pump has been shown to be responsive to cellular concentrations of sodium ions and that the activity of this pump utilizes a substantial proportion of the cell's ATP production, which varies with the genotype, physiological state, hormonal status, environmental conditions and diet (Milligan and McBride, 1985; Jessop, 2000).

Studies in cattle have shown that sodium pump activity accounted for 40% of total oxygen consumption and was elevated in young, as compared to older animals, by feed intake and exposure to cold, though breed was found to have little effects (Milligan and McBride, 1985). Further studies by McBride and Milligan (1985) with cattle showed that Na^+, K^+-ATPase activity increased from 35% in non-lactating or end of lactation cows to 55% during the peak of mid-lactation. The same authors have shown with sheep that the activity of the sodium pump accounted for 28%, 50% and 61% of total oxygen consumption at feeding levels of zero, maintenance and twice maintenance, respectively (McBride and Milligan, 1986). In the same study, it was shown that Na^+, K^+-ATPase activity was lowest (at 18% of total) for starved sheep and highest (45% of total) for animals at peak lactation.

Other published literature evidence on sodium pump energetics varies widely and include reports by Milligan and summers (1986) and Jessop (1988). An *in vivo* study by Swaminathan et al. (1989) using ouabain injection in guinea pigs showed a 40% reduction in whole-body metabolic rate. According to Jessop (2000), the maintenance of a cellular gradient of sodium and potassium ions in the face of both passive and active influx of Na^+ into the cell is energetically expensive. This need for the establishment of such gradient has been shown to originate from many sources including the need for the cell to use positive charges to balance negative charges due to proteins and nucleic acids across the cell membrane to maintain electroneutrality (Jessop, 2000).

The major aspects of total energy expenditure by the Na^+, K^+-ATPase system have been reviewed by Jessop (2000) and includes costs due to sodium/proton exchange, coupled nutrient transport systems, sodium ion leakage, cell volume regulation and energy production. Energy expenditure due to sodium/proton exchange has been attributed mainly to the effects of hormones (Pouyssegur et al., 1988) and weak acids (Park et al., 1992). On the other hand, cellular ion leakage/flux, especially across the lipid bilayer of the cell membranes has been shown to be highly energetic and is proportional to membrane surface area, difference in membrane ionic concentration and the permeability of the particular membrane to the ion in question (Brand et al., 1991).

Coupled transport of nutrients across the intestinal brush border has been extensively covered in a previous section. Not only are these systems highly energetic and ion-dependent but also show considerable variabilities within different animal systems (Mattews, 2000; Russell and Gahr, 2000). In effect, variations in energy expenditure for cellular transport may partly explain differences in efficiency of utilization of metabolizable energy. The possibility of metabolizable energy usage due to physical transmembrane transport activity may lead to greater insights into components of cellular energy metabolism. Such information may be very useful in the assessment of differences in energy use between animals of differing genotype (Jessop, 2000).

Body protein and fat accretion is the net result of the dynamic processes of synthesis and breakdown. Studies have shown that in both humans and domestic animals, as high as 20-25% of body protein can be broken down per day in early life and this slows with age to about 1-2% daily in adults (Rathmacher, 2000). A similar trend probably exists for body adipose tissues (Drackley, 2000; Kopecky et al., 2001). This however depends on a number of factors including plane of nutrition, stress, disease, hormonal effects, physical activity and inactivity as well as overall intracellular energy levels. Protein synthesis (translation) is a highly coordinated and energetic process with the involvement of more than 100 macromolecules (DNA, mRNA, tRNA, rRNA, activating enzymes, and protein factors). The process of synthesis is regulated by both the amount of RNA and the rate of translation. The energy cost of the overall synthetic process together with possible post-translational modifications may involve considerable energy expenditures in the form of ATP.

Protein breakdown involves the hydrolysis of an intact protein to amino acids and may be lysosomal and non-lysosomal. A second system of degradation is the ubiquitin-proteasome error-eliminating process, which involves the use of cytoplasmic peptidases to eliminate proteins that contain translational errors (Mitch and Goldberg, 1996). An additional cytoplasmic system of protein degradation involves the calpain-calpastatin Ca^{2+}-dependent autoproteolytic breakdown of myofibrillar proteins (Reeds, 1989). This system also requires utilization of high levels of energy in the form of ATP (Rathmacher, 2000).

On the other hand, lipogenesis refers to the synthesis of fatty acids (excluding esterification of fatty acids to glycerides). Adipose tissue is the main site of fat storage in non-lactating ruminant livestock. The mammary gland of lactating animals actively synthesizes fatty acids. Normal pathway for fat synthesis occurs in the cytosol and involves a sequential cyclical process in which acetyl units (usually from acetyl-Coenzyme A) are successively added to lipogenic primers (usually acetyl-CoA but also 3-OH butyrate). The acetyl units are derived from glucose (from glycolysis) or from acetate through rumen fermentation of dietary carbohydrates in ruminants. Fat tissue metabolism contributes greatly to the regulation of the body's energy balance. The amount of body fat reflects the balance between rates of accumulation and breakdown of triacylglycerols occurring in adipocytes. This accumulation depends on the extraction of lipids from circulation mediated by lipoprotein lipase (Kopecky et al., 2001).

In farm animals, the utilization of ME above maintenance depends on the partitioning of energy between protein and lipid synthesis and the respective efficiencies of synthesis. Early published evidence by the research group of Milligan and McBride (Gill et al., 1989) using mathematical representations of the energy requiring processes of protein turnover in the tissues of lambs to predict the relative contributions to ATP expenditure at different growth rates indicated that protein turnover accounted for 19% of whole-body ATP expenditure. Of this total protein turnover energy cost, 25-27%, 21-26%, 23-26% and 13% was attributable to turnover in GIT, muscle, skin and liver respectively. According to van Milgen and Noblet (1999), although on a weight basis, the lean mass of farm animals is much greater than the adipose tissue mass, the energetic density of adipose is about three to four times greater than that of lean tissue.

The energy density of lipid is typically taken to be 9.5 kcal/g and that of protein as 5.7 kcal/g. Consequently, less energy is required to deposit 1 kcal of energy as protein than as lipid. Due to the greater energy density of lipids, it is estimated that approximately 12 kcal is needed to deposit 1 g of lipid, whereas approximately 9.6 kcal is needed to deposit 1 g of protein (van Milgen et al., 2000). However, the synthesis of amino acids as proteins requires at least 5 ATP molecules, with an efficiency of 60% (implying that approximately 20 ATP molecules are required). It has been suggested that one of the major causes of this difference is the higher rates of protein turnover, especially in splanchnic tissues, compared to lipid turnover. Also, body fat is stored with little or no water whereas ~300% of the total weight of body protein is stored water.

Estimates by Emmans (1997), incorporating the energy cost of transporting amino acids into cells and then joining those amino acids into proteins may be approximately 12 Mcal/kg of protein retained. These estimates possibly did not consider the energy expenditure involved in transcription, translation, and post-processing modifications, which are essential processes in protein synthesis. On the other hand, Estimates by Emmans (1997) argue that when lipid is retained, there is a cost involved in the synthesis of the lipid molecules and also an amount of energy that is being stored as lipid. The energetic cost of lipid retention was therefore 13.5 Mcal/kg retained. Recent work done by van Milgen and Noblet (1999) shows similar results, but these authors also argue that the response of both protein and lipid deposition to energy supply may be highly variable, especially due to breed differences, and these differences may greatly affect how the energy supply is partitioned between protein and lipid deposition.

Literature evidence also suggests that energy expenditure associated with protein metabolism, especially involving non-essential amino acids may be very high but which is not directly associated with protein synthesis (Reeds et al., 1998). Key amongst these is glutamic acid, of which very little dietary supplements escape intestinal metabolism. Because glutamic acid is the only amino acid that can be synthesized by mammals through reductive amination of a ketoacid, it is the ultimate nitrogen donor for the synthesis of other non-essential ones. Accordingly, the synthesis of glutamic acid, and its product glutamine, involves the expenditure of high amounts of ATP. This gives evidence that non-essential amino acid synthesis might have a significant bearing on the energetics of protein synthesis, and hence, of protein deposition. In conclusion, several of the processes involved in the metabolic activities of the body are energetically expensive. The relative contributions of different tissues to total body energy expenditure should therefore be considered in the selection of target candidates for gene expression studies as well as candidates for genetic association studies to identify those genes that contribute to variation in feed intake and energetic efficiency in farm animals. In Genearal, it appears that the liver, GIT and CNS are by far the most energetically expensive organ systems.

2.6 Mapping of genes controlling quantitative traits in cattle

2.6.1 Introduction

Significant improvements in animal performance, efficiency and carcass and meat quality have been made over the years through the application of standard animal breeding and selection techniques based on genetic evaluation of animals for various quantitative traits. However, such classical animal breeding techniques require several years of evaluation of performance records on individual animals and their relatives and are therefore very time consuming and expensive. Molecular genetic tools in the form of genetic polymorphisms associated with specific traits of economic importance have been developed as a result of advancements in molecular, statistical and computational biology (Schwerin, 2001). These molecular technologies may complement classical animal breeding techniques and allow relatively easy and more efficient selection and breeding of farm animals with an advantage for an inheritable trait of growth rate, body weight, carcass merit, feed intake, feed efficiency, and milk yield and composition through marker assisted selection (MAS).

The use of MAS would be especially beneficial for those traits that have low heritability, are difficult or expensive to measure, are not directly measurable, or cannot be measured until the animal has already contributed to the next generation or is dead (and may be phenotypically but not genetic correlated to a trait that should not be increased; Spelman and Bovenhius, 1998). Marker-assisted selection may be applied through the information from polymorphisms in candidate genes that show associations with traits of interest (otherwise known as quantitative trait nucleotide, **QTN**) and QTL shown to be significantly associated with traits of interest. Several molecular tools and approaches as well as statistical and computational tools and techniques are available that can be employed to quantify the number(s), location(s) and effect(s) of QTN and QTL through the use of genetic markers.

A QTN may be defined as individual or groups of genetic polymorphisms, either of known or unknown chromosomal location and primary function, which shows significant association(s) with complex trait(s) of interest in a population. Several polymorphisms in candidate genes have been shown to be associated with economically relevant traits in cattle (Chrenek et al., 1998; Barendse et al., 2001; Ge et al., 2001; Grisart et al., 2002; Buchanan et al., 2002; Moore et al., 2003; Li et al., 2004; Nkrumah et al., 2005). On the other hand, a QTL is defined as the chromosomal location of individual or groups of genes, of unknown primary function, that show significant association(s) with complex traits of interest. In beef cattle, QTL have been detected for disease tolerance (Hanotte et al., 2003), fertility and reproductive performance (Kirkpatrick et al., 2000), body conformation (Grobet et al., 1998), birth weight and growth performance (Davis et al., 1998; Casas et al., 2003; Kim et al., 2003; Knealand et al., 2004), and carcass and meat quality (Keele et al., 1999, Casas et al., 2000, MacNeil and Grosz, 2002, Casas et al., 2003, Kim et al., 2003, Moore et al., 2003, Li et al., 2004). Several more QTL have been detected in dairy cattle that are not considered in this article. The objective of this section is to examine some of the principles and procedures used in mapping QTL in cattle and provide an overview of mapped QTL in beef cattle.

2.6.2 *Characteristics of genes influencing quantitative traits*

Genes are the fundamental basis for the structure and functioning of every organism and their analysis will eventually lead to the understanding of the genetic control of economically important traits. The segregation of alternative forms of a gene at multiple loci is the cause of much of the genetic variance component of the phenotypic variability in quantitative traits. According to Liu (1997), genes are the fundamental units of inheritance or segments of DNA on genomes and include structural genes with enzymatic regulatory functions in biosynthesis and regulatory genes that direct the functioning of other genes.

However, unlike genes that follow a typical Mendelian pattern of inheritance, QTL are more of statistical entities instead of biological ones and their effects can only be ascertained with given significance thresholds (Paterson, 1997). Genetic effects on quantitative traits are influenced or caused by certain characteristics of the gene, which can broadly be classified into the distributional properties of genes, interactive properties of genes and pleiotropic effects of genes.

With regards to the distribution effects of genes, it is widely known that mapping QTL involves extricating genetic signals from many sources of effects that are widely distributed in the genome (Paterson, 1997). One major hypothesis of gene effects distribution (the infinitesimal hypothesis) argues that genetic variation in quantitative traits is caused by very large number of genes with very small and equal allelic effects that cannot be estimated individually (Lynch and Walsh, 1998). Another model (major genes model) presupposes that distribution of allelic effects is nearly exponential and a few genes have large effects and cause much of the phenotypic variation and very large numbers of genes have very small effects on quantitative traits (Long et al., 1998). Analyses of marker associations with quantitative traits often reveal a number of QTL, with a few of large effects, and many more of smaller effect, on quantitative traits similar to an exponential distribution (Paterson, 1997; Hayes and Goddard, 2001).

Also, candidate gene molecular polymorphisms with large effects have been shown to be consistent with exponential distribution of gene effects (Orr, 1999). This genetic property can affect the results of QTL experiments because, according to Bost et al. (2001), low precision in estimating QTL positions may be due to factors such as undetected QTLs with small effects and overestimation of the effects of detected QTL, small sample sizes, low heritabilities, linkage disequilibrium between QTL, as well as epistatic interactions and unequal distribution of QTL effects. In general however, QTL with very large effects map to the smallest genomic regions and polygenes with small effects cover a wider region of the genome (Nuzhdin et al., 1999). This has implications for stating and testing hypotheses in QTL mapping, as the methodology may not only yield false positives, but may also inflate the phenotypic variance of the estimated effects of detected QTLs.

Certain genetic and non-genetic properties of genes influence their effects on complex traits. The major genetic properties are additive gene action, dominance gene action and genotype-by-genotype interactions (epistatic interactions). The major non-genetic properties include interactions with the sex of the organism (genotype by sex interactions) and interactions with the external environment (genotype by environment interactions) (Li, 1997). Additive genetic effects are due to the contribution of a fixed value differently to the overall phenotype by the different forms of a gene. That is, they contribute to the additive genetic variance of the population. Dominant gene actions result in the masking of the contribution of recessive alleles to the overall variance in the population. Epistatic interactions occur at both the genetic and molecular levels and influence certain developmental or biochemical pathways leading to the modification of expression of the homozygous or heterozygous states of the trait (Mackay, 2001). This may result in biases in the estimation of the main effects of QTL. Epistatic interactions between loci have been postulated to exist for QTL (Falconer, 1989), and can be synergistic (complementary gene action) when phenotype at the particular locus is enhanced by the interaction of genes at one or more loci. It may be antagonistic, in which case the phenotype of one locus is suppressed in the presence of genotypes at another locus (Routman and Cheverd, 1997).

Epistasis may also be in the form of duplicate gene effects, dominance epistasis and dominance suppression. Though a discussion of the molecular basis of epistasis is beyond the scope of this review, it is worthwhile stating that epistasis can occur between QTLs, between QTL and modifying loci, and between complementary loci (Mackay, 2001). Though the existence of different epistatic interactions has been well established at the molecular and physiological levels, the characterization of their effects on complex traits is very challenging and very little contribution of epistasis to variation in quantitative phenotypes has been established so far (Li, 1997). There seems to be presently no approaches in QTL mapping in cattle that takes into consideration epistatic effects.

Epistasis has obvious implications for both detecting and using QTL in breeding programs. For instance, the existence of interactions prevents the meaningful isolation of the effect of individual QTL and such QTL would not contribute, as predicted, to the selection pressure. Casas et al. (2001) identified a region on bovine chromosome 8 with an epistatic interaction with the myostatin gene that affects fat depth. Though the inability to confirm QTL observed in one population in another population is largely attributed to the non-segregation of alleles of the QTL in the latter population, it may also be due to the epistatic effects of QTLs. If the QTL is the result of epistatic interactions of two genes, then differences in genetic structure of different populations results in difficulties in confirming QTL in another population (Li, 1997).

Genotypes by sex interactions usually result in significant variations in terms of sexual dimorphism for the trait in question. The differences may be due to sex linkage producing the same effects in males and females or may be due to autosomal loci with different effects in opposite sexes. In this case the extent of sexual dimorphism in the quantitative trait depends on the magnitude of the interactions (Mackay, 2001). Some examples of such interactions are the effects of spontaneous and induced mutations and molecular polymorphisms on morphology of Drosophila, body size in mice (Templeton, 1999; Vaughn et al., 1999), and human disease susceptibility (Vieira et al., 2000). Genotype by environment interactions primarily lead to changes in the phenotypic value for a particular genotype. They are detected when the same genotypes are evaluated in multiple environments using near isogenic lines, recombinant inbred lines, selfed progeny of F2 individuals or molecular markers at a candidate loci (Mackay, 2001). The interactions may exist even if trials in multiple environments produce the same phenotypic effects. This property of QTLs has significant implications for many agriculturally important traits, especially in livestock production, when environmental changes have considerable impacts on productivity.

The pleiotropic effects of genes result from the fact that most genes controlling quantitative traits usually have more than one effect. Pleiotropy is the production by a single gene of two or more apparently unrelated phenotypic effects. That is, when one gene (or gene cluster) has multiple effects on multiple traits (Falconer and Mackay, 1996). Pleiotropy has been recognized as the major cause of the genetic correlation (together with linkage disequilibrium) observed among quantitative traits, which leads to correlated response to selection. Some genes can cause positive pleiotropy and others negative pleiotropy and the balance determines the genetic correlation of the two characters. It is the most important property of a quantitative trait gene as the effects from all loci contributing to the phenotypic variation in the trait determine the magnitude of genetic variation segregating for the trait, as well as the relative contributions of additive, dominance, and epistatic variances (Mackay, 2001).

Thus, variation in quantitative traits is caused by the segregation at multiple QTL with individual large or small effects that are sensitive to the effects of other genes, different sexes and external environmental factors. QTL genotypes cannot be determined from just the variations in phenotypes in a given population, but deductions about the net effects of all loci affecting the trait can be made by using a model that partitions the total phenotypic variance into components attributable to additive, dominance and epistatic variance, pleiotropy, genotype-environment interactions, genotype-sex interactions, and other external variances, such as age of individual or its dam (Mackay, 2001).

2.6.3 Molecular techniques and tools employed in QTL mapping

The main purpose of analyzing the genome is to determine the molecular basis of genetic variations in phenotypic traits in particular species through the identification and typing of DNA sequence variations (Nickerson et al., 2001). Many molecular techniques and tools have been developed and employed over the years for the analyses of the genomes of many prokaryotic and eukaryotic organisms. The major molecular genetics and other techniques that have been applied to genomic analysis include genetic mapping, physical mapping, radiation hybrid mapping, and DNA sequencing (Wagner, 1994). Though several of these techniques may be indirectly essential in the detection of QTL, a detailed discussion of each is beyond the scope of this paper. The major aspects of genetic mapping are genetic map construction, comparative mapping, and identification of the genes of interest. The primary step to performing an experiment to detect QTL involves the generation of genetic maps covering the entire genome of the organism with relative dense collections of polymorphic markers and location of all markers and protein coding genes (Bovenhuis et al., 1997).

Genetic map construction involves the determination, by means of an abstract model, of the linear arrangement of genes and genetic markers on the chromosomes in the genome of an organism. The process involves pairwise linkage analysis of observed and expected frequencies of possible genotypic classes (based on the analysis of homologous recombination during meiosis) using maximum likelihood or chi-square approaches

(Hildebrand et al., 1994). Markers are then grouped into different linkage groups based on the recombination fraction, significance level of the fraction and the number of chromosomes. Markers in the same linkage group are then ordered by determining the relative locus of each marker to produce a genetic linkage map. Comparative mapping involves the comparisons of the genetic maps of relatives in the same species or different species based on the theory of inter- and intra-specific synteny conservation. This does not only aim at information transfers from one species to another but also assists in the understanding of genome evolution and structure (Womack and Kata, 1995).

Though a complete description of these procedures is not included here, it is worthwhile to mention that, perhaps, the most important tool discovered in the analysis of the genome is the development of an *in vitro* method for the selective amplification of a short region of a DNA molecule, otherwise known as the polymerase chain reaction (**PCR**) (Mullis et al., 1986). PCR provides a simple and less expensive technique for the amplification (duplication or reproduction) of even small amounts of particular regions of the DNA. By way of repeating cycles of three reaction steps (strand separation, primer annealing to template DNA and synthesis of new strands), each occurring in less than two minutes, amplification into millions of copies of a piece of chromosome can be achieved.

DNA polymerase (Taq polymerase, a heat-stable polymerase isolated from the bacterium *Thermus aquaticus*) can characterize, analyze and synthesize any specific piece of DNA or RNA, even on extremely complicated mixtures, by seeking out, identifying, and duplicating bits of genetic material from blood, hair, or tissue specimens (Mullis et al., 1986). Basically, a PCR reaction requires a piece of the template molecule (DNA or RNA to be copied) and two single-stranded DNA primers (informing sequence that anneal, or bind by complementary base pairing to the template and is responsible for the beginning and reversion of the reaction). The mixture also contains free deoxyribonucleoside triphosphates (dNTPs), heat-stable DNA polymerase (an enzyme that catalyzes the synthesis of DNA strand complementary to the target sequence) and a reaction buffer to facilitate primer annealing and optimize enzymatic function (Mullis et al., 1986).

The role of genetic markers in the detection of QTL cannot be overemphasized. In cattle, QTL mapping has been facilitated by the development of relatively dense genetic maps covering all chromosomes (Barendse et al., 1994; Kappes et al., 1997; Ihara et al., 2004; Snelling et al., 2005). Present genetic marker coverage of the bovine genome include more than 5,000 marker positions spanning a region of over 3,000 cM. This has been as a result of the collective efforts of many international collaborators. Three types of genetic markers have been used in genomic analysis, namely morphological markers, protein based markers and DNA based markers (Paterson, 1997). Several criteria are used to judge the suitability of a genetic marker for genomic analysis. These may include locus specificity, degree of heterozygosity, polymorphism information content (**PIC**), level of biological resolution, cost effectiveness, rapidity of technique for use in high throughput analysis, sample size requirements (preferably PCR based), repeatability and reproducibility of technique across different laboratories, and the availability of the technique in a range of species (Nickerson et al., 2001).

Morphological marker analysis (morphometric analysis) employs phenotypic measurements of categorical traits such as shape, colour, size, etc., that can be used as reliable indicators for specific genes (Summers and Medrano, 1994). Two main types of protein marker analyses are known, namely allozyme analysis (isozyme analysis) and analysis based on sodium dodecylsulfate polyacrylamide gel electrophoresis (SDS-PAGE). A relatively large number of morphological markers have been studied in livestock species and may be due to simple point mutations or may not have a simple genetic basis. They often define and identify expressed genes and are easy to observe. However, it is difficult to have a high number of them segregating in a single population or few populations and they are generally known to be relatively poor indicators of underlying genetic variations (Paterson, 1997). Isozyme analysis is simple, relatively inexpensive and requires little preliminary work, as it does not require species-specific DNA. However, lack of high numbers of potential isozyme markers limits its application to only highly polymorphic systems, with little or no application in QTL mapping and map-based cloning in farm animals.

Protein marker analysis based on SDS-PAGE depends on the denaturation of proteins with sodium dodecylsulfate and gel electrophoresis followed by staining and visualization. Allelic differences are detected by means of differences in polypeptide size due to differences in length of coding regions or post-translational modification of proteins (Burrow and Blake, 1997). There are also cytogenetic markers (analyzed by chromosome banding techniques) and immunologic markers (analyzed by antigenic cross activity using enzyme-linked immunosorbent assay - ELISA). ELISA is cheap and uses easily accessible technology but it is limited in the number of markers that may be produced and it is relatively unpolymorphic, and thus low in information content (Burrow and Blake, 1997).

DNA markers comprise small regions of DNA exhibiting sequence polymorphisms in different individuals within a species (Vaiman, 1999). Two basic techniques have been employed in the detection of DNA based markers, namely fragment detection by nucleic acid hybridization such as used in the analysis of restriction fragment length polymorphisms (**RFLPs**) (Botstein et al., 1980) and sequence amplification using the polymerase chain reaction such as those methods used for analyzing minisatellites, microsatellites, sequence tagged sites (**STSs**), and expressed sequence tags (**ESTs**) (Mullis et al., 1986). The discovery of RFLPs marked the shift from protein marker analyses to DNA marker systems (Botstein et al., 1980). RFLP analysis employs the detection of individual variability in DNA sequence by means of restriction endonucleases (obtained from microbial cells), which cut genomic DNA into small fragments according to specific recognition sequences (usually six base pairs, bp).

The technique involves separation of DNA fragments of various sizes through gel electrophoresis followed by transfer of fragments to nylon membranes (southern blotting) and visualization of specific fragment sizes separated by length. Visualization uses radioactive or chemiluminescent probes exposed to an X-ray film (Drinkwater and Hetzel, 1991; Montgomery and Crawford 1997). Differences in restriction enzyme cut sites due to point mutations, insertions, deletions, translocations and duplications is the basis for the detection of RFLPs (Paterson, 1997). A number of RFLPs have been detected in cattle and there are some applications of RFLP techniques in mapping the bovine genome (Womack and Kata, 1995; Hetzel et al., 1997). A modification of the standard method of electrophoresis in RFLP analysis, denaturing gradient gel electrophoresis (**DGGE**) makes it possible to detect point mutations between restriction sites by separating samples on polyacrylamide gels containing a gradient of urea. This technique has been employed to map bovine chromosome 7 (Urquhart et al., 1996) and detect allelic polymorphisms for the bovine growth hormone releasing gene (Ge et al., 1999), as well as typing BoLA-DRB3 alleles (Aldridge et al., 1998). RFLP markers are co-dominant, and scoring is simple due to the low copy of probe numbers. However its application requires a complementary DNA (cDNA) or genomic library of the appropriate species.

The development of the polymerase chain reaction occurred alongside the discovery of a new type of molecular marker, which, unlike RFLPs, employs multiple copy polymorphisms due to allelic variations in the number of repetitive elements. This opened up the chance to map multiple, highly polymorphic markers in different species (Montgomery and Crawford 1997). The genomes of most mammalian species are replete with many classes of repetitive elements such as long interspersed nuclear elements (**LINEs**) and short interspersed nuclear elements (**SINEs**), which are terminated by particular types of tandem repeats known as satellite DNA (Vaiman, 1999). LINEs and SINEs have not received a wide application in mapping genes in livestock due to the difficulty in separating the large number of DNA fragments produced during analysis. On the other hand, the tandem repeat elements have been highly useful in dissection of the genomes of most organisms.

There are two major kinds of tandem repeats namely, variable number of tandem repeats (**VNTRs** or minisatellites) and simple sequence repeated polymorphisms (**SSRPs** or microsatellites) (Vaiman, 1999). Minisatellites are regions of DNA with polymorphisms in the number of repeated nucleotide sequences (usually 10 to 100 base pairs long) and analyzed by means of probes known as minisatellite monomers. They are usually genotyped by southern blotting by means of restriction enzymes that cut the sequences flanking the marker region. Minisatellites are organized into families of related sequences. The distribution of minisatellites varies among species, but they are known to be widely distributed in the bovine genome (Georges, 1991). Minisatellites can be detected using both hybridization approaches and the polymerase chain reaction. However, minisatellites are usually not amenable to PCR. Also, relatively fewer markers have been characterized in domestic species and the cost of high throughput development is high (Georges, 1991). Minisatellites have therefore received little applications in genomic analysis.

Microsatellite markers or single sequence repeat polymorphisms are a kind of highly polymorphic tandem repeats consisting of segments of genomic DNA of about one to six base pairs used for genetic analysis through PCR amplification systems. They were first discovered when eukaryotic genomic DNA was subjected to isopycnic cesium chloride density gradient centrifugation (Litt and Luty, 1989; Webber and May, 1989). The high degree of polymorphisms, informativeness, abundant genome-wide distribution, specificity of primers, the ease of detection, and the ability to evaluate multiple markers of known location make microsatellite markers highly useful in genomic map construction in livestock species (Smith and Smith, 1993; Stein et al., 1996; Montgomery and Crawford 1997). This has especially been boosted by the development of multiplexed microsatellite PCR reaction systems, which can be analyzed by automated DNA sequencing systems.

Microsatellite analysis is performed by amplification of genomic DNA using pairs of specific primers flanking tandem arrays of repeat sequences. Their high and remarkable informativeness is due to the ability of the tandem repeat sequences to contract during DNA replication. Differences in the number of repeats can be reliably distinguished, and the variants are inherited as alleles at a single genetic locus (Montgomery and Crawford 1997). There are several reports cited in the literature about the early application of bovine microsatellite markers in mapping QTL (Moore et al., 1994; Stone et al., 1999). High throughput microsatellite genotyping may be achieved presently through the Applied Biosystems 3730 DNA Analyzer with the accompanying software, GeneMapper® v3.7.

According to Sonstegard et al. (2001), the difficulty to fully automate techniques for genotyping microsatellite markers led to the development of a new class of molecular markers known as single nucleotide polymorphisms (**SNPs**). The SNPs are polymorphisms due to single nucleotide substitutions, insertions, deletions, translocations and duplications. They are highly abundant in the genomes of humans and mice, as well as cattle and other mammalian species. Single nucleotide polymorphisms are biallelic with an average of 30 - 50% heterozygosities in most populations. There are systems available for mining and identifying SNPs from EST databases (Sonstegard et al., 2001). Several techniques can be used to genotype SNPs including single stranded conformational polymorphism (**SSCP**) assays, allele specific oligonucleotide (**ASO**) assays, SNP discrimination by electronic dotblot assay on semiconductor microchips (dynamic allele specific hybridization or DASH); amplification refractory mutation systems (**ARMS**); minisequencing and analysis on DNA chips or by polyacrylamide gel electrophoresis; minisequencing and analysis using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (**MALDITOF**). Currently, there are several chemistries available for genotyping SNP. Of these, the Illumina® BeadStation 500G delivers impressive assay conversion rates and accuracy (Oliphant et al., 2002; Shen et al., 2005).

There are other molecular markers such as sequence tagged sites (also known as amplicon length polymorphisms (**ALP**) or specifically amplified polymorphisms (**SAP**) and EST specific markers. Other markers are produced from restriction landmark genome scanning (**RLGS**), amplification of undefined elements (e.g. amplified fragment length polymorphisms or **AFLPs**) (Sasazaki et al., 2001), and random amplification of polymorphic DNA (**RAPD**) (Horvath and Medrano, 1994) These may have received higher levels of genetic applications in analyzing the genomes of other organisms, especially plants, but have had limited applications in livestock species (Montgomery and Crawford 1997).

2.6.4 *Experimental design considerations in QTL mapping*

Mapping QTL involves the use of linkage information from genetic markers to search for associations with trait phenotypes. This process requires the use of populations that are segregating for the QTL and the markers used. Thus, specific experimental designs based on specific resource populations are required. The major experimental design issues in QTL mapping are related to the selection of a mapping population and the determination of the scheme for mating. The objective of the study is usually the determining factor in the selection of parents, mating design, and the type of markers used. Sufficient molecular and genetic variation for the traits of interest must exist in the parents of the mapping population. There are three major design possibilities used in QTL mapping, namely crosses between divergent inbred lines to produce an inbred population (similar to recombinant inbred lines), crosses between outbred populations, and crosses within outbred populations.

The first two methods are collectively referred to as experimental crosses and the mapping population may be either a backcross, an F2 cross, or an advanced intercross (F3, F4, etc.) produced from divergent populations (Visscher et al., 1996). There are other population types under these categories such as the use of double haploids, repeated backcross populations and testcross populations. The backcross population is produced from a cross between either two inbred strains (inbred lines method) or outbred strains (outbred populations method) to produce the first filial (F1) generation. The F1 animals are crossed with one of the parental strains to produce the mapping population that are phenotyped for the trait.

The F2 cross (second filial generation) from outbred populations is produced by crossing (selfing) individual F1 animals. The F1 animals in both crosses would be heterozygous for every chromosomal position (marker position) that the parental strains differ. There are two putative QTL genotypes per locus in the backcross animals and three genotypes per locus for the F2 population. Though backcross populations are simpler to analyze compared to F2 crosses, the latter provides more opportunity and information to examine the genetic structure and architecture of the QTL and provides more power than backcross analysis (Liu, 1998). The use of inbred lines is the most efficient methodology in QTL mapping because it maximizes the linkage disequilibrium between marker and QTL genotypes and increases the mapping resolution.

In most livestock populations, especially cattle due to the generation interval, it is too cumbersome and expensive to establish experimental animal crosses (except when such crosses already exist). Thus, a common approach is to use crosses within outbred populations. Mapping populations based on variations within outbred lines aim at exploiting existing family structures and may be made up of half sib or full sib individuals (progeny design) or higher generation families (great grand- and grand progeny designs) (Weller et al., 1990; Uimari et al., 1996, Moody et al., 1997). These designs are currently existent in commercial herds. Families from the same line (breed) can be expected to originate from a limited number of common ancestors. In these designs, proven male animals, say bulls, have many male offspring, which in turn have many progeny. Genotypes are collected on a number of grandsires and their half-sib offspring and phenotypes may be collected on the half-sib offspring themselves (progeny or daughter design) or on a group of progeny from each half-sib (grand progeny design).

In the progeny design differences in the mean phenotypic or breeding values of the offspring inheriting alternative forms of marker alleles from the heterozygote sire is evidence for the existence of QTL. The granddaughter design has generally been employed in dairy cattle and phenotypic data is in the form of daughter yield deviations (**DYD**) for the trait of interest computed for each son based on the performance of its daughters (Weller et al., 1990). Differences in the mean yield deviation values of the offspring inheriting alternative forms of marker alleles from the heterozygote grandsire is evidence for the existence of QTL. Though the procedure could be extended to include full-sib family structures as long as family sizes are large enough, QTL mapping using full-sib families is yet to be attempted in cattle. The grand progeny designs are often used for genome scanning as it reduces the number of markers that are required to be typed to obtain a QTL detection power similar to the progeny design (Weller et al., 1990; Moody et al., 1997). These methods are however, less powerful in QTL detection as the QTL alleles may not be preferentially fixed in the parental populations and there is more uncontrollable variability in the other parents of half-sibs.

Other methods for mapping that may be employed for within outbred population QTL analysis, which help to reduce the amount of genotyping required include selective genotyping (individuals from the phenotypic tails of the population are genotyped for markers flanking the confidence interval of the putative QTL; Darvasi and Soller, 1992) and selective DNA pooling or bulk segregant analysis (Carleos et al., 2002) of half sib or full sib animals. In addition, procedures such as the sib-pair analysis and the transmission disequilibrium test which were originally designed for QTL mapping for both discrete and quantitative traits in humans are now also being adapted for QTL analyses in farm animals. Generally, QTL mapping using within line variations in commercial populations largely ignore the genetic relationships between animals in different lines, and this is bound to result in some bias in the estimation of QTL effects. A methodology known as the variance components QTL mapping has been developed which would incorporate direct genetic relationships between sires and dams (Meuwissen and Goddard, 1996). The QTL effect is modeled as a random effect and the model used would account for all genetic relationships at the QTL by modeling the identical-by-descent (**IBD**) probabilities between alleles of individuals derived from marker genotypes (Meuwissen and Goddard, 1996). The procedure is similar to BLUP and would provide genotypic breeding values for all individuals. These methodologies would however not be discussed in detail in this paper.

2.6.5 Statistical and computational considerations in QTL mapping

Generally, the methods used for QTL mapping employs either linkage analysis or exploitation of population-wide linkage disequilibrium (**LD**). In linkage analysis, evidence for the existence of a QTL is based on the genetic linkage between the QTL and a marker. Generally, individuals are marker-genotyped to determine the series of linked alleles (haplotypes) on the chromosome, and the association between marker and QTL depends on the frequency of recombination (Meuwissen and Goddard, 2000). The approach is to find those markers with the lowest recombination frequencies with the QTL, and therefore have the highest effects. The maximum mapping resolution obtainable from linkage analysis in a given pedigree corresponds to the interval between the two nearest flanking crossovers. The major principle of QTL mapping based on population-wide LD is that, in a large non-inbred, random mating unselected population, markers and QTL are expected to be in linkage equilibrium across the population (Knott et al., 1996). QTL detection based on markers requires linkage disequilibrium (or a correlation in gene frequencies) between marker and QTL (Womack and Kata, 1995). In livestock species, LD exists even between distant loci (Haley, 1999), and could extend as far as 20 cM (Farnir et al., 2002). Linkage disequilibrium is a useful method if there are only two alleles at the QTL, since the presence of more alleles at the QTL makes detection of LD very difficult (Meuwissen and Goddard, 2000).

Principally, if a QTL were linked to a marker locus, then there would be a difference in mean values of the quantitative trait among individuals with different genotypes at the marker locus. The closer the distance between the QTL and the marker, the greater is the difference in trait phenotype between the different marker genotypes. The maximum difference in mean values occurs when the marker genotypes coincide exactly with the QTL (Mackay, 2001). Linkage disequilibrium is not only caused by physical linkage but also by drift, selection, crossing (hybridization), or by the influence of founder animals (Mackay, 2001).

2.6.6 *Alternative approaches to QTL mapping*

Basically, the procedures for QTL mapping aim at testing specific statistical hypotheses by way of statistical models (genetic models of the traits and models for the relationship between conceivable QTL and genetic markers). There are single- and multiple-QTL models. Single-QTL models are models without QTL interaction (Liu, 1997). In this case, though many markers may be tested, only one or two may be directly related to the putative QTL and the other markers are present to control genetic background effects and sampling errors. Multiple models are based on the fact that the tests for the hypothesis are not independent among marker loci due to linkage and possible gene interactions (that is epistatic effects) (Liu, 1997). Different methodologies have been used to map QTL based on populations from experimental crosses or commercially existing family structures. The different methodologies, in the order of complexity, include single marker association, interval mapping, composite interval mapping, multiple interval mapping and automatic construction of multi-QTL models by Bayesian methods (Manly and Olson, 1999).

Single-marker analysis tests for associations between trait values and the genotypes at the marker locus for either backcross, F2, half-sib, or full-sib animals. The test may involve the one target marker locus and a number of background marker loci (that have already been shown to be associated with the trait and therefore lie close to the QTL) using the general linear model for ordinary least squares estimation. According to Knott et al. (1996) and Haley et al. (1994), single-marker analysis wastes information and may be potentially biased in the estimation of QTL location due to confounding of QTL genotypic means and QTL positions. The approach also requires the separate estimation of the location and effect of each QTL detected, but estimation of the effect is a combination of QTL position and actual effect, which is non-independent (Korol et al., 1996). Also, individuals with missing marker genotypes have to be excluded from the analysis.

An alternative method of QTL analysis to single marker analysis is simple interval mapping (**SIM**) developed by Lander and Botstein (1989) and employed in chapter 8 of this research project. The method requires the prior construction of a marker linkage map. Simple interval QTL mapping approaches have been shown to possess more power than single-marker QTL analysis and provide more accurate estimates of the position and effects of QTLs (Lander and Botstein, 1989; Haley and Knott, 1992). The method evaluates the association between the trait values and the expected contribution of a hypothetical QTL at multiple analysis points between each pair of adjacent marker loci and assumes the presence of a single QTL. The genotypes of animals from informative parents for the flanking markers are used to estimate the likelihood of the expected QTL genotypes based on the genetic distance. When the marker genotype at the flanking marker for an individual is not available, the next flanking marker can be used instead.

The major advantages of SIM over single marker analysis include the fact that the method provides a curve which indicates the evidence for the QTL location. The estimates of the QTL effects are more accurate. The method makes proper allowances for incomplete marker genotype data because only the closest informative flanking typed marker is considered for calculating the QTL probabilities for each individual. If an individual is missing the marker genotype for the flanking marker, the method moves to the next flanking informative marker for which genotype data is available. Finally, the method allows for the inference of QTLs to positions between markers (Manly and Olson, 1999). It has however been noted that the application of interval mapping techniques is not straightforward, especially for non-inbred populations. The technique is also computationally demanding. Additionally, since markers may not be completely heterozygous for haplotypes, there may be variations in informativeness from interval to interval depending on type of flanking markers. Also, the number of QTL cannot be resolved and QTL positions are not well resolved (Knott et al., 1996). Finally, for crosses involving multiple families, only a few families may be informative for the QTL.

As an alternative to interval mapping, Jansen and Stam (1994) proposed composite interval mapping (**CIM**) methods, which combine maximum likelihood interval mapping methods with multiple regression. The aim of CIM is to evaluate the possibility of a target QTL at multiple analysis points across each inter-marker interval. This approach reduces bias in estimates of QTL positions by decreasing the intra-marker-class phenotypic variation through the use of cofactors by means of background markers. If the cofactor and the target interval are not linked then the analysis becomes more sensitive to the presence of a QTL in the target interval. However, if they are linked, then the cofactor helps to separate the new QTL from other linked QTL near the cofactor (Zeng, 1994). This principle has been extended to apply to multiple traits and has now been refined into a method known as multiple interval mapping (Kao et al., 1999). These approaches are however unable to distinguish between significant effects caused by single QTL or effects caused by clusters of multiple QTL with small effects (Vilkki et al., 1997). The major problem with CIM is the decision of what markers to be used as cofactors in the analysis. As a solution to this problem, an extension of CIM known as multiple interval mapping (**MIM**) for multiple QTL was proposed (Kao et al., 1999). The method allows the inference of QTL position between markers and makes proper allowance for missing genotype data and can allow interactions among QTLs. This approach incorporates information from all markers to provide relatively fast methods for analysis of the entire genome (Knott et al., 1996).

This approach may also be used in conjunction with some of the other approaches listed above. The principle behind this approach is that, given relative genome positions through a linkage group, the probability of an offspring inheriting one or the other of its sire's haplotypes at the locus is conditional on its marker haplotype. Therefore, by inferring the marker haplotypes inherited by each progeny from its sire through permutation or other re-sampling technique, sire haplotypes for the markers can be reassigned (De Koning et al., 2001). Programs have been developed that use stepwise regression or simple regression of each marker to identify eligible background markers as cofactors depending on the strength of their association with the trait (Zeng et al., 1999).

2.6.7 Testing for the presence of a QTL

Two general methods have been used to estimate QTL effects namely, least squares (regression or ANOVA) (Haley and Knott, 1992; Jansen, 1993) and maximum likelihood (**ML**) estimation using either restricted error maximum likelihood (REML) (Patterson and Thompson, 1971) or the expectation-maximization (**EM**) algorithm (Lander and Botstein, 1989, Zhang and Hoeschele, 1998). Least squares methods are the simplest methods used in QTL detection. The model assumes QTL genotype effects to be fixed, and regresses the probability of the putative QTL genotypes given marker information on the phenotypic data. However, least squares methods cannot distinguish between effects tightly linked to the marker and those that are loosely linked (Knott et al., 1996). Another problem with least squares methods is the inherent assumption that trait phenotypes are normally distributed. Trait phenotypes may be correctly assumed to be normally distributed if QTL genotypes were known unequivocally. However, given only marker genotypes, trait distributions comprise mixtures of overlapping normal distributions in which the underlying environmental effects are not normal. Least squares linear approximations, though very effective in practice, do not sufficiently account for the differences in trait distributions across marker genotypes (Jansen, 1993).

The ML approach models the distribution of trait values as a mixture of overlapping normal distributions and uses iteration, which are generally slower than regression procedures. The methods initially assume arbitrary starting values for QTL strength and residual effects from which better estimates are then made after several steps of iteration and convergence at the ML value. A third method of estimation of QTL effects is the moments estimation method (Darvasi and Weller, 1992) but this method is seldom used. The statistical technique used for testing the significance of the hypothesis of a marker-trait association may be a Student's t-test, an F-test (Markel et al., 1996), the logarithm of odds (**LOD**) score (Lander and Botstein, 1989), a log likelihood ratio (G) test (Haley and Knott, 1992), or a non-parametric test (Kruglyak and Lander, 1995). The lod score is computationally similar to the G-statistic with the exception that the latter is computed as the natural logarithm and the former is computed using the base 10 logarithm. In addition, the lod score is interpreted using the concept of odds ratio whilst the G-statistic is the probability of occurrence of the data given the null hypothesis and uses a theoretical Chi-square distribution.

The different mapping methodologies have found wide applications in mapping QTL in several livestock species. However, these approaches have been found to be less versatile, QTL location estimates are generally poor (especially if only one marker is used), location of QTL could be either side of the marker, and the method is computationally intensive (Knott et al., 1996). These mapping techniques are continually being refined to ensure accurate estimation of QTL positions and effects, and to increase their ease of use.

As an alternative to the above methods of QTL mapping, several efforts have been made to implement QTL mapping models that allows the automatic construction of models using Bayesian inference approaches (Jansen, 1996, Uimari et al., 1996). Bayesian methods for QTL mapping are implemented through Bayesian Markov Chain Monte Carlo (**MCMC**) algorithms (Thaller and Hoeschele, 1996) using the so-called Gibbs sampler (Janss et al., 1995) and are computationally more efficient and intensive. The methods allow the automatic consideration of multiple QTLs models, QTL positions, and QTL strength. This approach always gives exact densities of variance components unlike other methods that have unknown distributions for small data sets (Jansen, 1996). According to Hoeschele (1994), the use of the Gibbs sampler as an MCMC relies on a numerical integration that iteratively generates samples from the full conditional densities of all the unknowns such as marker and QTL allele frequencies, number of QTLs, additive, dominance, and epistatic effects of QTLs, QTL and marker genotypes, linkage parameters, marker and QTL map positions, fixed effects, and polygenic variances, and may be used to correct marker genotyping errors.

2.6.8 Statistical problems in QTL mapping

Two major issues are currently very contentious in QTL mapping namely, the question of statistical power and the problems associated with multiple testing. Statistical power relates to the number of animals with phenotypic data and the number of markers required for detecting the QTL at an appropriate resolution (otherwise known as the power of the analyses) (Liu, 1998). Power of QTL mapping depends on the type of cross, the size of the QTL effect, the population size, marker density in the region of the QTL and the significance level. The QTL effect can be strong (> 20% of the trait variance), moderate (may only be detected with reasonable population sizes, irrespective of the power) and weak QTL (< 1% of the trait variance). Differences in experimental power may lead to overestimation or underestimation of QTL effects and may determine whether the results of the study appear to be statistically significant. To avoid overestimation of the effects of detected QTL, maintain the predetermined significance threshold, and avoid poor repeatability (due to low statistical power and inability to detect all QTL), a more stringent approach has to be employed in declaring QTL as significant or significant levels have to be empirically estimated (Churchill and Doerge, 1994).

The second and perhaps, the most contentious issue in QTL mapping studies is the problem of multiple testing. Generally, QTL mapping involves the determination of the probability of obtaining a test statistic (LOD score, G-value, t-value, F-value, regression coefficient, etc.), which is larger than that observable if there were no QTL. Very large test statistics give small probabilities indicating that the null hypothesis of no QTL is false or a very rare false positive has occurred. In statistical testing, it is usual to reject the null hypothesis of no linkage if the probability of obtaining observed results under the null hypothesis is less than a standard threshold, typically 5% (Liu, 1998). However, QTL mapping uses a number of repeated analyses, which includes the number of markers tested, the number of small genome positions tested (in the case of interval mapping) and the number of traits tested. When many tests are performed addressing the same issue, such as linkage of a trait across a genome, we expect that fully 5% of the tests performed will produce observations significantly different from the null hypothesis at the 5% level even when there is truly no linkage present (Liu, 1998).

As results of this, the number of tests conducted are not independent because some markers are linked, epistatic interactions occur, and some of the traits are genetically correlated with each other (Liu, 1998). Thus the probability level for a test statistic to be significant used for testing one trait at one marker location is not appropriate for forming an overall significance threshold. Adjustments to the threshold must be made in line with the number of multiple testing carried out. Several approaches have been proposed to adjust the significance level for a test statistic. Lander and Botstein (1989) and Knott and Haley, 1992) as well as others propose a Monte Carlo simulation method to build a distribution of significance levels under the null hypothesis that is appropriate for particular genome sizes. Lander and Kruglyak (1995) proposed the suggestive, the significant, and the highly significant levels of QTL detection corresponding to one false positive per genome scan, genomewide Type 1 error rate of 0.05 and genomewide Type 1 error of 0.01, respectively.

Churchill and Doerge (1994) proposed the permutation test to obtain empirical threshold values that are appropriate for each particular study. The approach involves the randomization of the phenotypic data only (keeping the genotype data intact) and performing the analysis 1,000 to 10,000 times to determine the maximum test statistic. By comparing the actual test statistic given the data to the ones obtained from the permutation test, the 100 (1 – α) percentile in the distribution of the maximum test statistic is taken as the experiment-wise critical test statistic. This approach is most advantageous as it provides a threshold value that is customized to the particular experiment. However, permutations tests are highly time consuming and may be impractical in some situations. According to Manly and Olson (1999), p values from permutation tests need not be determined with high precision and could be determined from the standard error of the empirical p value, which is equal to $[p(1-p)/N]^{0.5}$, where p is the threshold level and N is the number of permutations.

Whereas the permutations test method of Churchill and Doerge (1994) is generally accepted to most researchers, some of the earlier methods are based on the procedure for Bonferroni correction and are not generally acceptable due to its over-stringency (Perneger, 1998). False positives or negatives in QTL mapping may be due to insufficient power to detect the QTLs or unreasonable statistical thresholds, which may become obvious in replicated studies. In general, QTLs detected by one study but not by another should not be ignored because such variability in findings may not only be related to Type I or Type II error rates but by other factors such as the genetic structures of the populations used as related to differences in allele frequencies, especially of those genes that seem to have epistatic effects on some other traits.

2.6.9 Specific computer software for QTL mapping

According to Paterson (1997), QTL are unlike genes that follow a typical Mendelian pattern of inheritance, as they are more of statistical entities instead of biological ones and their effects can only be ascertained with given significance thresholds. Quantitative trait loci analyses involves many repeated analyses in one task and the test statistics lack one standard distribution and are generally complex in nature (Liu, 1997). Over the years, several computer software packages for QTL mapping have been developed by statistical geneticists. Liu (1998) and Manly and Olson (1999) provide comprehensive reviews of most of the available software, the contact details of the authors/owners, and the abilities and weaknesses.

Examples of some of the software packages include MapMaker/QTL (Lander et al., 1987; Lincoln et al., 1992), QTL Cartographer (Basten et al., 2001), MAPQTL™ (Van Oijen and Maliepaard, 1996), Map Manager QT (Manly et al., 2001), and QGENE™ (Tanksley and Nelson, 1996). Others include PLABQTL (Utz and Melchinger, 1996), MQTL (Tinker and Mather, 1995), Multimapper (Sillanpää, 1998), the QTL Café, and Epistat (Chase et al., 1997). There are other software such as WINQTLCART (Wang et al., 2004), MULTICROSSQTL (Rebai et al., 1997), MQREML, MQAREML, MPLGIB, NQTLGIB (specially designed for QTL mapping in outcross pedigrees (Hoeschele et al., 1998) and QTL Express which is generally based on the regression methods described by (Haley and Knott, 1992). Most of these vary widely in user interface and operating systems they support. Most of them are public domain programs except QGene and MapQTL. Almost all of the above programs can perform single marker associations, simple interval mapping and composite interval mapping using backcross, intercross (F2), outbred populations as well as in recombinant inbred lines.

Generally, all these programs have the following Characteristics: 1) the interface is not user-friendly (except for commercial software and QTL express), 2) user-support is limited, 3) there is limited flexibility for user-defined models; but 4) speed of analyses is fast for those models that the programs can handle (Manly and Olson, 1999). Most of the software packages also require known linkage map positions for running, and some of these programs come with such software packages such as MapMaker/EXP, GMENDEL, PGRI, MapManager and JoinMap, etc., (Liu, 1998). Compared to all these packages, several authors recommend QTL mapping based on SAS (Liu, 1998) or other general statistical programs such as Genstat, and most of the software packages listed above can apparently be implemented in general statistical packages. The major advantages of using general statistical packages such as SAS include the fact that the software are commercially available, user interfaces are friendly, user support is available with or without charge, and the user specifies the models appropriate for his/her situation. Single marker association using SAS can be implemented easily by most animal scientists as models are generally ordinary or generalized least squares.

Liu (1998) outlines the approaches for simple and composite interval mapping using linear and non-linear regression approaches which can easily be followed. Recently, a SAS version of QTL mapping software (QTL-by-SAS) has been launched (Xu et al., 2003). According to these authors, many scientists, especially quantitative geneticists and animal and plant scientists are familiar with SAS, but are not necessarily so with C++ and other computer languages and will feel more comfortable to run a SAS program than to run a C++ program. This is simply because they understand the codes and can easily modify the programs to fulfill their own needs. In addition, a SAS version of the QTL mapping program will bring the statistical problems of QTL mapping to the attention of the SAS developers and stimulate their desire to develop a SAS procedure for QTL mapping (PROC QTL).

2.6.10 Gene mapping studies for economically relevant traits in beef cattle

According to Fries (1993), gene mapping is the first step in genome analysis that leads to a complete understanding of the genetic control of economically important traits in livestock species. There are two major approaches to obtaining genomic information for the genetic improvement of livestock, namely the candidate gene approach (uses knowledge of the physiology or biochemical affinity of genes with the nature or expression of the trait) and the QTL mapping approach (based on statistical inferences on the detection of associations between genes and polymorphic sequences of DNA in the same chromosome). Most of the identified polymorphisms in candidate genes in cattle affect the coding sequences of the specific genes and usually form the molecular basis for many monogenic traits or disorders.

A review of such characteristics is provided by Schwerin (2001) and examples include bovine leukocyte adhesion deficiency (BLAD) (results from an A/G transition at the β_2-integrin locus on bovine chromosome, **BTA1**) (Kehrli et al., 1990), Pomp's disease (results from a point mutation in the acid *a*-glycosidase gene on BTA19 (Schwerin, 2001), congenital hyperthyroidism (resulting from a C/T transition in the TG gene on BTA14) (Ricketts et al., 1987), roan, white heifer disease (resulting from a transition in the mammary gland factor, STAT5 gene on BTA19) (Seitz et al., 1999), muscle hypertrophy or double muscling trait (resulting from deletions/insertions in the myostatin, MH gene on

BTA2) (Grobet et al., 1998) as well as several point mutations in the κ-Casein (CASK) gene on BTA6 which confer improved cheese making properties (Medrano and Aguillar-Cordova, 1990).

Polymorphisms in certain candidate genes have also been associated with various quantitative traits in cattle. These include polymorphisms in the TG5 gene associated with increased marbling (Barendse et al., 2001), polymorphisms in the leptin gene associated with carcass fatness, feed intake, milk yield (Buchanan et al., 2002; Buchanan et al., 2003 Liefers et al., 2002, 2003; Nkrumah et al., 2004), and serum leptin concentration (Nkrumah et al., 2005), variants in the calpastatin gene associated with toughness or tenderness (Bindon, 2002), and polymorphisms in the growth hormone gene associated with differences in growth rate, carcass weight and meat yield (Chrenek et al., 1998). Others include growth hormone receptor polymorphisms and weaning and carcass weight (Hale et al., 2000), insulin-like growth factor-I (IGF-1) polymorphism associated with weight gain (Ge et al., 2001), myogenic factor (MYF5) gene polymorphisms with growth rate and body weight (Li et al., 2004) as well as polymorphisms in the pituitary-specific transcription factor (PIT1) polymorphism associated with body depth, angularity, and rear leg set (Renaville et al., 1997).

One of the most successful stories in candidate gene studies was reported by Grisart et al (2002) in which a non-conservative lysine to alanine substitution (*K232A*) in the bovine *DGAT1* gene was shown to be a causative quantitative trait nucleotide underlying a QTL affecting milk fat composition, which was previously mapped to the centromeric end of bovine chromosome 14. This polymorphism has subsequently been shown to affect marbling score in beef cattle (Thaller et al., 2003). Though the candidate gene approach is very useful, as the identity of the actual gene that affects the trait may ultimately be known, the approach is often unsuccessful and many genes that do not have known functions would be ignored as potential candidates and their important effects would be missed (Kirkpatrick, 1999). Many factors may account for the limited success in the use of the candidate gene approach, including the polygenic nature of many quantitative traits in farm animals, appearance of mutations, possibility that the putative QTL is, in fact, a marker near the QTL allowing recombination, possibility of interactions between genes in different loci which can modify the expression of genes in different populations, and the fact that most genes do not show quantitative variation which greatly affect their function despite their involvement in the physiology of the trait (Kirkpatrick, 1999).

Unlike the search for polymorphisms in candidate genes that affect traits of economic importance, QTL mapping involves the use of genetic markers to detect regions of the genome that account for a significant variation in the trait of interest. The main advantage of QTL mapping, compared to the candidate gene approach is that a search could be performed to provide indications of chromosomal fragments which show observed significant variations in a particular trait without any prior knowledge about the physiological or biochemical functions of the QTL. Great progress has been made in identifying QTL for various production traits in cattle, most of which are in dairy cattle (Georges et al., 1995; Ashwell et al., 1997; Vilkki et al., 1997; Arranz et al., 1998; Zhang et al., 1998), but also including several important traits in beef cattle. A summary of the findings of some recent QTL mapping studies in beef cattle are presented in Table 2.1.

Lien et al. (2000) (using outbred animals and MIM) and Kirkpatrick et al. (2000) (using a granddaughter design with both CIM and MIM) detected QTL for twinning rate and ovulation rates, respectively. Davies et al. (1998) detected QTL for birth weight in a half-sib tropical beef cattle herd on *Bos taurus* (BTA) chromosomes 5, 6, 14, 18 and 21 with effects of between 0.39 to 0.82 units of within sire standard deviation. Grosz and MacNeil (2001) detected a QTL for birth weight on BTA 2 with an effect of 0.64 residual standard deviations using a half-sib progeny of Hereford X composite cross. Also, Machado et al. (2003) detected a QTL for birth weight on BTA 5 suing a half-sib population of Canchim (Charolais x Zebu) cattle. Li et al. (2002) detected a region that is negatively associated (0.79 SD) with birth weight on BTA 5 using identity-by-descent haplotype analysis in a commercial (Angus) line of beef cattle. Casas et al. (2000) also detected a QTL for birth weight on BTA 6 in a Belgian Blue x Piedmontese population segregating an inactive form of the myostatin gene. Most of these studies also detected QTL for birth weight that shows suggestive significance. Other studies reporting QTL for birth weight include Stone et al. (1999) and Kim et al. (2003).

Machado et al. (2003) detected a QTL for yearling weight breeding values on BTA 5 using a half-sib population of Canchim (Charolais x Zebu) cattle at a distance 0.1 cM from the IGF-I locus. Li et al. (2002) detected regions associated (0.79 SD) with preweaning (0.60 SD) and postweaning ADG (0.65) on BTA 5 using identity-by-descent ordinary least squares haplotype analysis in a commercial (Angus) line of beef cattle. Casas et al. (2000) also detected a QTL for yearling weight on BTA 6 in s Belgian Blue population segregating an inactive form of the myostatin gene. Several QTL for carcass and meat quality traits have been detected in cattle. These include carcass weight (Elo et al., 1999; Casas et al., 2000, 2003; Kim et al., 2003), backfat thickness (Casas et al., 2000, 2001 and 2003, Moore et al., 2003 and Li et al., 2004), marbling score (Casas et al., 2001, 2003; MacNeil and Grosz, 2002), LM area (Casas et al., 2003), retail product yield (Stone et al., 1999; Casas et al., 2003) and dressing percentage (Stone et al., 1999; Casas et al., 2000). There are also QTL for meat quality, but these have generally been carried out on shear force measurements (Warner-Bratzler) (Keele et al., 1999, Casas et al., 2000).

Generally, the definition of QTL intervals by standard genome scans and statistical analysis (such as those reviewed here) result in very poor resolution, with support intervals ranging from 20-30 cM (Sonstegard et al., 2001). Several factors may account for this including the phenotypic variance of the QTL to the overall spread of the phenotype, the spacing of the genetic markers used in the analysis, the pedigree size of the experimental population, the type of design (outbred, F2, or backcross) used, the existence of more than one locus under the QTL peak contributing an effect, the distance between these linked loci, and lack of accuracy suffered by most QTL mapping algorithms (Moore and Nagle, 2000). Considering such a wide range, the mapping resolution would be seriously insufficient for any efficient application to animal breeding.

High-resolution mapping may therefore be used to determine the approximate location of the QTL, by successively reducing the size of the region flanking the QTL. Several approaches have been developed to localize QTL after initial detection from general mapping studies. The most appropriate approach for livestock species relies on the exploitation of historical recombination events (through identical-by-descent haplotype sharing methods) through the identification of animals in which a recombination event has occurred between nearby flanking marker(s) and QTL by extending QTL analysis to include ancestral generations of animals in the pedigree (Darvasi, 1998). These approaches have been used by Li et al. (2002, 2003, 2004) to reduce the support intervals of the reported QTL for certain growth and carcass traits in cattle.

According to Georges (1999), the fastest, most inexpensive, and most recommended approach to fine mapping for improvement of map resolution is to saturate the area surrounding the QTL position with markers. This could be achieved by generating genotypes from markers that are known to be near the potential QTL position, based on reference linkage maps. The only limitation to this approach is the availability of enough informative markers across the region of interest in the genome. With the linkage maps currently available for cattle, marker densities are of the order of 0.5-3.0 cM on average per chromosome and several collaborative efforts are underway to more than double this density across the entire bovine genome (Sonstegard et al., 2001).

Another powerful experimental strategy employed to localize QTL to a small genomic distance for possible positional cloning is the identification of candidate genes by comparative mapping. The principle of comparative mapping is based on the overall evolutionary conservation of synteny (on the same chromosome) between species. Identification of these evolutionary break points between conserved syntenic groups in different species allows the extrapolation of marker information from the gene-rich highly developed human and mouse maps to the lower density cattle map (Womack and Kata, 1995). This approach does not only represent potentially the most efficient way to increase the density of the most accurate markers in the vicinity of QTLs, but also identifies critical biochemical pathways for further investigation and manipulation. Conserved chromosome segments are estimated to be an order higher than 10 cM on average between man and livestock species. These and several other approaches are expected to be used extensively in the coming years to assist in documenting several QTL in cattle and other farm animals.

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Table 2.1. Overview of QTL studies for some economically relevant traits in beef cattle

Trait	Source	Breed	Design	BTA	Location, cM
Twinning	Lien et al., 2000	Nordic cattle	Commercial half-sib	5	75-85
Ovulation	Kirkpatrick et al., 2000	Swedish Friesian	Selective genotyping	5	100-120
Ovulation	Kirkpatrick et al., 2000	Swedish Friesian	Selective genotyping	19	50-80
Birth Wt.	Davis et al., 1998	Tropical herd	Half-sib	5, 6	70-110
“	Davis et al., 1998	Tropical herd	Half-sib	14	0 and 42
“	Davis et al., 1998	Tropical herd	Half-sib	18	100-130
“	Davis et al., 1998	Tropical herd	Half-sib	21	4 (0-30)
“	Grosz & MacNeil, 2001	Hereford	Half-sib	2	114
“	Machado et al., 2003	Charolais x Zebu	Half-sib	5	82.9
“	Li et al., 2002	Angus	Commercial line	5	0 -30
“	Casas et al., 2003	Brahman	Half-sib	5, 6, 21	42-68
“	Stone et al., 1999	Brahman	Half-sib	1	95-135
“	Kim et al., 2003	Brahman x Angus	Full-sib	2, 6	126, 1
Carcass Weight	Elo et al., 1999	Finnish Ayrshire	Grand daughter	23	10-40
“	Casas et al., 2003	Brahman	Half-sib	29	45-58
“	Casas et al., 2000	Belg. Blue & Pied	Half-sib	6	50-60
“	Casas et al., 2003	Brahman	Half-sib	18	11-38
“	Casas et al., 2003	Brahman	Half-sib	10	0-30
“	Kim et al., 2003	Brahman x Angus	Full-sib	23	14
Backfat	Casas et al., 2001	Belg. Blue & Pied	Half-sib	4	15-40
“	Casas et al., 2003	Brahman	Half-sib	7	44-71
“	Casas et al., 2003	Brahman	Half-sib	14	0-22

Table 2.1. continued.

Trait	Source	Breed	Design	BTA	Location, cM
Backfat	Casas et al., 2003	Brahman	Half-sib	2	21-60
"	Casas et al., 2000	Belg. Blue & Pied	Half-sib	5	40-80*
"	Casas et al., 2000	Belg. Blue & Pied	Half-sib	14	10-20*
"	Casas et al., 2001	Belg. Blue & Pied	Half-sib	8	0-16*
"	Li et al., 2004	Angus	Commercial	5	65.4-70
"	Li et al., 2004	Angus	Commercial	6	varied
"	Li et al., 2004	Angus	Commercial	19	varied
"	Li et al., 2004	Angus	Commercial	21	46.1-53.1
"	Li et al., 2004	Angus	Commercial	21, 23	45.1-50.9
Marbling	Casas et al., 2001	Belg. Blue & Pied	Half-sib	3	40-90
"	Casas et al., 2001	Belg. Blue & Pied	Half-sib	8	0-20
"	Casas et al., 2003	Brahman	Half-sib	23	21-42
"	MacNeil & Grosz, 2002	Hereford	Half-sib	2	112-132
LM area	Casas et al., 2003	Brahman	Half-sib	5	38-66
"	Casas et al., 2003	Brahman	Half-sib	6	0-26
Product yield	Stone et al., 1999	Brahman	Half-sib	2	30-40
"	Casas et al., 2003	Brahman	Half-sib	9	63-92
Dressing %	Casas et al., 2000	Belg. Blue & Pied	Half-sib	5	60-75
"	Stone et al., 1999	Brahman	Half-sib	5	30-80
"	Stone et al., 1999	Brahman	Half-sib	5	45-85
Shear force	Keele et al., 1999	Brahman	Half-sib	15	17-40
"	Casas et al., 2000	Belg.Blue & Pied	Half-sib	5	40-80
"	Casas et al., 2000	Belg. Blue & Pied	Half-sib	29	55-70

CHAPTER 3

Genetic and Phenotypic Relationships of Feed Intake and Different Measures of Feed Efficiency with Growth, Ultrasound, and Carcass Merit of Composite Cattle [1]

3.1 Introduction

The goal of efficient and productive feeding of animals, within economic and environmental constraints, is to provide essential available nutrients for maintenance and production with minimal excesses and losses. The high cost of feeding beef cattle coupled with the potential environmental impacts of beef production (Herd et al., 2002) implies that the feed efficiency of beef cattle is an economically relevant trait. More recently, the availability of automatic feed intake monitoring technology (e.g., based on radio-frequency technology) has enabled the accurate measurement of the individual intakes of groups of animals housed together over reasonable time periods, which then allows the estimation of genetic and phenotypic (co)variance components for indices of feed intake and feed efficiency. Efficient beef cattle production involves a complex summation of appropriate levels of available feed inputs and product outputs over a range of different production systems involving animals at different developmental stages. Thus, several indices have been proposed for determining the energetic efficiency of beef production (Archer et al., 1999).

Traditionally, feed efficiency has been expressed in terms of feed conversion ratio (**FCR**), or its inverse (gross feed efficiency, **GFE**), and it is measured as the ratio of feed consumed to gain in weight. Feed conversion ratio is highly correlated with growth and it is greatly confounded with growth rate and maturity patterns of different animals (Archer et al., 1999). Using FCR as an index of feed efficiency could result in substantial increases in the feed requirements of the cow herd, which would then negate the gains made from the high growth rate of young animals, resulting in negative impacts on the overall production system efficiency (Barlow, 1984; Dickerson, 1978).

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[1] A version of this chapter has been published. Nkrumah et al., 2004. J. Anim. Sci. 82: 2451–2459.

Alternative measures of beef cattle feed efficiency include residual feed intake (**RFI**, Koch et al., 1963), maintenance efficiency (Webster et al., 1974), cow-calf efficiency, partial efficiency of growth (**PEG**, Kellner, 1909; Archer et al., 1999), relative growth rate (**RGR**, Fitzhugh and Taylor, 1971) and Kleiber ratio (**KR**, Kleiber, 1947; Bergh et al., 1992). Residual feed intake is defined as the difference between an animal's actual feed intake and its expected feed intake based on its BW and growth rate over a specified period. Recently, RFI has been shown to have greater potential as an index of feed efficiency for beef cattle (Liu et al., 2000; Herd and Bishop, 2000; Arthur et al., 2001a), RFI. Differences in RFI may however be associated with differences in carcass fatness (Richardson et al., 2001; Basarab et al., 2003), carcass leanness (Herd and Bishop, 2000; Arthur et al., 2001b) and meat quality (McDonagh et al., 2001), which may not be entirely desirable to the beef cattle industry; genetic associations of RFI with other measures of carcass merit are generally unknown, with exception of phenotypic relationships reported by Basarab et al. (2003) and Nkrumah et al. (2004).

Very little is currently known about how RFI compares to other proposed measures of efficiency such as FCR and PEG (Arthur et al., 2001a) in terms of relationships to feed intake, growth rate, and especially carcass merit. In addition, several recent studies have focused on establishing the genetic and phenotypic relationship between RFI computed from phenotypic regression (**RFIp**) with BW, ADG, and feed intake (Arthur et al., 2001a, b; Crews et al., 2003a; Herd and Bishop, 2000; Schenkel et al., 2004). However, Kennedy et al. (1993) and Crews (2005) have reiterated that, though the phenotypic independence of RFIp with BW and growth may be forced, genetic associations may still remain; RFI computed from genetic regression (**RFIg**) has been proposed instead to ensure genetic independence between RFI and its component traits, though this has not been widely investigated. Recently, Hoque and Oikawa (2004) and Hoque et al. (2005) have reported genetic and phenotypic relationships between RFIp and RFIg in Japanese Black cattle. The present study was conducted to compare different measures of feed efficiency (RFIp, RFIg, FCR, and PEG) in terms of the genetic and phenotypic relationships with growth, feed intake, and ultrasound and carcass merit of crossbred composite cattle.

3.2 Materials and methods

3.2.1 Animals and Management

Growth, feed intake, ultrasound, and carcass merit data were collected on composite beef steers over three yr (November 2002 to June 2005). The animals were produced from a cross between Angus, Charolais or University of Alberta Hybrid Bulls and the University of Alberta's experimental hybrid dam line. The Dam line was produced from crosses among three composite cattle lines, namely Beef Synthetic 1 (BS1), Beef Synthetic 2 (BS2) and Dairy X Beef Synthetic (DBS). Briefly, BS1 was composed of approximately 33% Angus and Charolais, about 20% Galloway, and the remainder of other beef breeds. The BS2 composite was made up of about 60% Hereford and 40% other beef breeds. The DBS was composed of approximately 60% dairy breeds (Holstein, Brown Swiss or Simmental) and approximately 40% beef breeds, mainly Angus and Charolais (Goonewardene et al., 2003). They were managed and tested for growth and feed efficiency under feedlot conditions at the University of Alberta's Kinsella Research Station using the GrowSafe automated feeding system (GrowSafe Systems Ltd., Airdrie, Alberta, Canada). All animals in the study were cared for according to the guidelines of the Canadian Council on Animal Care (CCAC, 1993).

Cows and heifers were bred in multiple sire breeding groups on pasture and the sire of each calf was later determined in a parentage test using a panel of bovine microsatellite markers. The animals weighed 353.0 (SD = 61.3) kg and were 252 (SD = 42) days of age at the beginning of testing. Two tests made up of approximately 80 animals per test were conducted each year. Each year, animals were randomly assembled into two contemporary test groups. Each animal was identified by means of a plastic tag located in the left ear. All animals had been vaccinated for bovine viral diarrhea and clostridial diseases and treated with a pour-on parasiticide before entry into the test. In yr one, steers were fed free choice a backgrounding diet of mainly alfalfa-brome hay with oats supplemented with corn grain and feedlot mineral supplement to promote a growth rate of just under 1.0 kg/d for approximately 30 d. This period was followed by a 30-d pre-test adjustment period in which the amount of corn in the backgrounding diet was adjusted up gradually to introduce the animals to the test diet and the feeding system. This was done to allow them to adapt to the diet and learn to feed from the test facility.

The test diets for the three years are presented in Table 3.1. The test diet in yr one was composed of 80.0% dry-rolled corn, 13.5% alfalfa hay pellet, 5% feedlot supplement (32% CP beef mineral supplement containing 440 mg/kg of monensin, trace minerals and vitamins) and 1.5% canola oil, supplying approximately 2.90 Mcal/kg of ME and 12.5% CP. In yr two and three, the same test procedures were used, but the test diet contained 64.5% barley grain, 20% oat grain, 9.0% alfalfa hay pellet, 5.0% beef feedlot supplement and 1.5% canola oil, supplying 14.0% CP and 2.91 Mcal/kg of ME. Corn was used in yr 1, instead of barley and oats, because of a feed barley shortage that particular yr. Prior to entry into the testing facility, each animal was fitted with a passive radio frequency transponder button (Allflex USA, Inc., Dallas/Fort Worth, Texas 75261-2261-2266, USA) encased in plastic ear tags at a position 5 to 6 cm from the base of the right ear with the button on the inside. The test facility was housed in a shed with one long side open to provide access to 10 feeding bunks. The animals were housed in a large outdoor pen with straw provided as bedding, small amounts of which may be eaten to promote rumination. Evidence from previous studies (Archer et al., 1997) in which straw were deliberately fed to animals (instead of as a bedding) indicate that the daily energy intake from straw provided as bedding would be insignificant. Individual animal intakes of straw were therefore not measured and excluded from calculation of intake.

Each yr, the test diet was sampled weekly and composited monthly. The composite samples were thoroughly mixed and sub-sampled for measurement of *in vivo* digestibility and proximate analysis carried out for dry matter, GE, fat, protein, ash, calcium, phosphorus, NDF and ADF (Table 3.1). The *in vivo* digestibility of the diet in yr one was determined using feed intake and faecal output data of three Suffolk sheep (107.7, SD = 6.1 kg BW) tested at the Metabolic Research Unit of the University of Alberta, Edmonton, Canada. Daily feed intake, orts and faecal output were measured on each sheep for 1 wk after they had been gradually brought to full feed (*ad libitum* intake with approximately 5% orts). Samples of the feed, orts and faeces for each animal were composited and frozen until analyzed. Samples were dried at 65°C and then at 100°C in a forced-air oven to a constant weight to determine DM. Feed intake was 1.70 ± 0.26 kg DM/d. The *in vivo* digestibility of the diet in yr two and three was determined using feed intake and faecal output data of 12 steers selected and used in digestibility and calorimetry trials (Nkrumah et al., 2006). The GE of the various samples was determined by bomb calorimetry using an automatically controlled Parr adiabatic calorimeter (Model 141, Parr Instruments Co., Moline, IL). The DE of the diet was computed from the GE of the feed and data obtained from the *in vivo* digestibility tests and converted to ME using the NRC (1996) equations. The DE and ME of the test diets obtained from digestibility trials were compared to those calculated using NRC (1996) tables and equations. In all yr the compositions determined by digestibility trials were similar. The NEm and NEg of the diets were also calculated using NRC (1996) equations.

3.2.2 Collection of Feed Intake, Growth, Ultrasound and Carcass Data

Feed intake was measured for each animal using the GrowSafe® automated feeding system, which has been validated and used previously (Basarab et al., 2003). Briefly, the system consisted of 10, 4000 E feed bunks, a data-logging reader panel connected to each feed node, a personal computer and GrowSafe® Data Acquisition and Analysis Software. Wireless communication (Model 4000 R/F) allowed for the transfer of data between the acquisition unit and a desktop computer in an office located approximately 100 m away. Daily feed intake for each animal as recorded by the GrowSafe® system was determined using specially customized software. Data collected (1-2% per test) on days when the automatic monitoring system failed to function due to power failure, mechanical problems or failure of a main computer board were excluded from all subsequent analyses for all animals.

Weight measurements of all animals were taken weekly (yr one) or fortnightly (yr two and three) and ultrasound measurements of 12/13th rib fat depth, LM area and marbling score were taken every 28 d. Ultrasound measurements were taken with an Aloka 500V real-time ultrasound with a 17 cm, 3.5-MHz linear array transducer (Overseas Monitor Corporation Ltd., Richmond, BC) using procedures detailed by Brethour (1992). At the end of the feed efficiency tests steers were weighed (harvest weight) and shipped to a commercial packing plant where they were slaughtered the following day and standard industry carcass data collected after a 24-h chill at -4°C.

3.2.3 Traits and their Derivations

Linear regression in SAS (SAS Institute, Inc., Cary, NC, version 9.1.3) of weekly or fortnightly BW measurements against time (d) was used to derive ADG, final BW and mid-test metabolic size (**MWT**, $BW^{0.75}$) for each steer. The total feed intake of each animal over a 70-d test period was used to compute the daily DMI. Each animal's FCR was computed as the ratio of DMI to ADG on test. The PEG of each animal (i.e., energetic efficiency for ADG above maintenance) was computed as the ratio of ADG to the difference between average daily DMI and expected DMI for maintenance (**DMIm**) (Arthur et al., 2001), where DMIm was computed according to NRC (1996). Residual feed intake was calculated both from phenotypic regression (RFIp, Arthur et al., 2001a) or genetic regression (RFIg, Crews et al., 2005; Hoque and Oikawa, 2004) of ADG and MWT on DMI. Test group (six levels) was included as an independent variable in the calculation of RFI. In each case, individual RFI was computed as actual DMI minus the expected DMI predicted from the appropriate phenotypic or genetic regression model.

Rate of gain and final ultrasound backfat (**UBF**), LM area (**ULMA**), and marbling score (**UMAR**) were predicted from regression equations of ultrasound measurements upon time (d) for each individual animal. Carcass traits were evaluated according to the Canadian beef carcass grading system (Agriculture Canada, 1992). Carcass weight (**CWT**) of each animal was determined as the sum of the weights of the left and right halves of each carcass. Carcass grade fat (**CGF**) and LM area (**CLMA**) were measured at the 12/13th rib of each carcass. Carcass marbling score (**CMAR**) is a measure of intramuscular fat and can be classified as 1 to < 2 units = trace marbling (Canada A quality grade, **QG**); 2 to < 3 units = slight marbling (Canada AA quality grade); 3 to < 4 units = small to moderate marbling (Canada AAA quality grade) and ≥ 4 units = slightly abundant or more marbling (Canada Prime). Lean meat yield (**LMY**) is an estimate of saleable meat and was calculated according to the equation, lean % = 63.65 + 1.05 (muscle score) - 0.76 (grade fat). Yield grade (**YG**) is the proportion of lean meat and is classified as Y1 = >59 %, Y2 = 54 to 58%, and Y3 < 54%.

3.2.4 Statistical Analyses

Performance, feed efficiency, and ultrasound records were available on 464 animals, and carcass merit records were available on 381 animals. The total number of animals including those without records but contributing pedigree information was 813. The animals in the study were primarily sets of paternal half-sibs, but some dams were used in multiple years on the same sires, making some animals full siblings. The animals were classified into high, medium and low RFIp groups based on ± 0.5 standard deviations from the mean, in order to determine the differences in performance and carcass merit among animals belonging to different classes of feed efficiency.

Differences in measures of feed efficiency, performance, ultrasound and carcass merit among steers in different classes of RFIp were determined using a SAS MIXED MODEL (SAS Institute, Inc., Cary, NC, version 9.1.3), which included fixed effects of RFIp group (high, medium, and low), breed of sire (Angus, Charolais, or Hybrid), test year (three levels), test group nested within year (two groups per year), and random effects of a classification variable of sire and dam of steer. Age of steer on test was included in the model as a linear covariate. Mean separation among RFIp groups for different test traits was carried out by least-squares using the PDIFF option in SAS. The PROC CORR of SAS was used to obtain Pearson partial phenotypic correlations adjusted for the linear (and sometimes quadratic) effects of age and the fixed effect of year.

Genetic (co)variances were obtained with the statistical software ASREML (Gilmour et al. 2000). A preliminary univariate animal model for each trait was fitted to obtain starting (co)variance parameters that were then fitted in subsequent restricted maximum likelihood (REML) bivariate analyses. Pairwise bivariate analyses were performed for each feed efficiency trait against the other test traits. The two-trait individual animal models used to estimate (co)variance components included fixed contemporary group effect, random additive genetic and residual effects, and linear covariate for age. Genetic variances and heritability estimates for any particular trait were calculated as the average value of the estimates from all pairwise bivariate analyses performed against all traits, while their SE were the medians of the SE estimates.

3.3 Results

Animals in the study averaged ADG = 1.46 kg/d, MWT = 90.22 kg$^{0.75}$, DMI = 10.45, kg/d, FCR of 7.36 kg DM/kg of gain (Table 3.2). Phenotypic residual feed intake averaged 0.00 kg DM/d and ranged from -2.73 kg/d (most efficient) to 2.86 kg/d (least efficient), a difference of 5.59 DM kg DM/d of feed. On the other hand, RFIg averaged -0.14 and ranged from -3.12 kg DM/d to 3.72 kg, a difference of 6.84 kg DM/d between the most and least efficient animals. Phenotypic variances for ADG, MWT, DMI, FCR, RFIp, RFIg, and PEG, respectively were 0.073, 109.6, 2.59, 1.59, 0.77, 1.02, and 0.0036, respectively. Corresponding heritability estimates were 0.59, 0.31, 0.54, 0.41, 0.21, 0.42, and 0.56, respectively. Heritability estimates for ultrasound backfat, LM area, and marbling score were 0.59, 0.39, and 0.75, respectively. Heritability estimates for harvest weight, carcass weight, grade fat, LM area, marbling score, lean meat yield, yield grade, and quality grade were 0.50, 0.33, 0.55, 0.45, 0.49, 0.63, 0.58, and 0.30, respectively.

The differences in feed efficiency, feed intake, growth, ultrasound, and carcass merit among animals in different RFIp groups are presented in Tables 3.3 and 3.4. It must be noted that animals with higher RFIp, RFIg, and FCR values are less efficient than animals with lower values. The difference in RFIp between high- and low-RFI RFIp animals was approximately 1.90 kg DM/d ($P < 0.001$). Phenotypic and genetic correlations among feed intake and different measures of feed efficiency with measures of growth, ultrasound and carcass merit are presented in Tables 3.5, 3.6, and 3.7. Generally, RFIp and RFIg were highly genetically and phenotypically correlated with each other ($r > 0.90$) and their corresponding phenotypic ($P < 0.001$) and genetic correlations with FCR and DMI were high. Least-squares means for RFIp, RFIg, FCR, DMI and ME intake were consistently higher ($P < 0.001$) for high-RFIp animals than for medium or low-RFIp animals ($P < 0.001$), and were higher ($P < 0.001$) for medium-RFIp animals than low-RFIp animals. Both RFIp and RFIg were strongly correlated phenotypically ($P < 0.001$) and genetically with PEG. Phenotypic RFI was higher for low-RFIp than medium- or high-RFIp animals ($P < 0.001$). Also, PEG was lower ($P < 0.001$) for medium-RFIp than low-RFIp animals.

In general, RFIp was phenotypically not related to ADG, MWT, final BW, or BW at harvest. There were no significant differences ($P > 0.10$) among different RFIp groups in LS means for ADG, metabolic BW, final BW, or harvest weight. Similar phenotypic correlations were obtained between RFIg and these traits, except with ADG where a weak but significant negative phenotypic correlation ($r = -0.21$, $P < 0.05$) was observed. On the other hand, ADG and metabolic BW were moderately genetically correlated with RFIp but the corresponding genetic correlations with RFIg were not different from zero. Generally, most of the phenotypic and genetic correlations among growth, feed intake and feed conversion ratio reported in this study were significantly different from zero. Daily DMI was moderately correlated genetically and phenotypically with FCR, but had strong genetic and phenotypic correlations with ADG and metabolic BW. Feed conversion ratio had strong genetic and phenotypic correlations with ADG, but had only a weak phenotypic correlation with metabolic BW; the genetic correlation of FCR with metabolic BW was not different from zero. Partial efficiency of growth had moderate and weak but significant phenotypic correlations with ADG and metabolic BW, respectively; the corresponding genetic correlations of PEG were strong with ADG and moderate with metabolic BW.

Phenotypic correlations of RFIp and RFIg with ultrasound and carcass backfat were moderate ($P < 0.01$). Compared to animals with low-RFIp, animals with high-RFIp had significantly higher rate of gain in ultrasound backfat ($P < 0.001$), final ultrasound backfat thickness ($P < 0.001$), carcass grade fat ($P < 0.001$), and average carcass backfat ($P < 0.001$; data not shown). Ultrasound backfat had moderate genetic correlations with RFIp but its correlation with RFIg was not different from zero. Carcass grade fat, on the other hand, was moderately genetically correlated with both RFIp and RFIg. The phenotypic correlation of RFIp and RFIg with ultrasound LM area, carcass LM area and ultrasound marbling score were not different from zero ($P > 0.10$). Carcass marbling score had low but significant ($P < 0.05$) phenotypic correlations with RFIp and RFIg. Consistently, there were no significant differences ($P > 0.10$) between different RFIp groups in rate of gain or final ultrasound LM area or ultrasound marbling score. Also, no differences were observed among different RFIp groups in carcass LM area or carcass marbling score. However, RFIp and RFIg had moderate positive genetic correlations with ultrasound marbling score, and low to moderate positive genetic correlations with carcass marbling score. On the other had, both measures of RFI had strong negative genetic correlations with ultrasound LM area and carcass LM area.

Carcass weight had low ($P < 0.05$) and moderate ($P < 0.01$) phenotypic correlations with RFIg and RFIp, respectively, but was genetically not correlated to both traits. No significant differences were observed among different RFIp groups in carcass weight ($P > 0.10$). Carcass lean meat yield ($P = 0.003$) and yield grade ($P = 0.02$) were different among animals with different RFI. Low-RFIp animals had higher lean yield and better yield grades compared to high-RFIp animals. Carcass lean meat yield had low negative phenotypic correlations with RFIp and RFIg. The genetic correlations of lean meat yield with RFIp and RFIg were moderate to high and negative. On the other hand, carcass yield grade had low ($P < 0.05$) and moderate ($P < 0.01$) positive phenotypic correlations with RFIp and RFIg, respectively. The corresponding genetic correlations of yield grade with RFI and RFIg were not different from zero.

Phenotypic correlations of FCR and PEG with ultrasound and carcass traits were not different from zero ($P > 0.10$), with the exception of the phenotypic correlations of PEG with ultrasound backfat ($r = -0.21$, $P < 0.01$) and carcass grade fat ($r = -0.14$, $P < 0.05$). The genetic correlations of FCR with ultrasound and carcass traits were generally different from, except with ultrasound marbling, carcass grade fat and carcass yield grade. Genetic correlations of PEG with ultrasound and carcass traits ranged from low to high and were generally different from zero except with ultrasound backfat and carcass weight. With the exception of the genetic and phenotypic correlations of DMI with lean meat yield, daily DMI had positive moderate to high phenotypic and genetic correlations with ultrasound and carcass traits. Lean meat yield had a moderate negative phenotypic correlation and a high negative genetic correlation with daily DMI.

3.4 Discussion

The results of the present study confirm the findings from several other studies (Arthur et al., 2001a, b; Crews et al., 2003a; Herd and Bishop, 2000; Robinson and Oddy., 2004; Schenkel et al., 2004), indicating the existence of considerable genetic variation in RFI, in younger cattle, to warrant its application in genetic selection to improve efficiency of feed utilization. Arthur et al. (2001a), using data on Angus bulls and heifers, reported heritability estimates of ADG, MWT, FCR, feed intake, and RFIp to be 0.28, 0.40, 0.39, 0.29, and 0.39, respectively. Additionally, Arthur et al. (2001b) reported heritability estimates on young Charolais bulls at 15 months of age for ADG, BW, feed intake, FCR, RFIp, and PEG to be 0.34, 0.37, 0.48, 46, 0.39, and 0.39, respectively. Thus, the heritability estimate of RFIp in our study was lower compared to the estimates by Arthur et al. (2001a, b) cited above. The genetic variance for RFIp in the present study (0.15) was slightly lower than reported (0.220) by Arthur et al. (2001a) but comparable to that observed (0.149) by Arthur et al. (2001b). On the other hand, the phenotypic variance of RFIp in this study (0.774) was higher than values reported by Arthur et al. (2001a, b; 0.58 and 0.55, respectively).

The heritability estimates obtained for the other traits in this study are generally moderate and similar to the estimates by Arthur et al. (2001a, b). Heritability estimates for feed efficiency and performance traits in similar studies (Crews et al., 2003a; Herd and Bishop, 2000; Koch et al., 1963; Renand and Kraus, 2002; Schenkel et al., 2004) were moderate and consistent with the findings of this study. The heritability estimate for genetic residual feed intake in the present study was higher than reported in Japanese Black cattle (0.25, Hoque and Oikawa, 2004; Hoque et al., 2005). The heritability estimates obtained from the various studies imply that selection for RFI has the potential to result in genetic change that is comparable to that obtainable from other moderately heritable traits in beef cattle (Crews, 2005). The heritability estimates for measures of ultrasound and carcass merit are consistent and within the range of values reported on carcass and ultrasound traits in general (Bertrand et al., 2001; Crews and Kemp, 2001; Crews et al., 2003b; Devitt and Wilton, 2001) or studies incorporating performance, feed efficiency and carcass traits (Arthur et al., 2001a; Robinson and Oddy, 2004; Schenkel et al., 2004).

Generally, most of the genetic and phenotypic correlations of feed intake and feed efficiency with each other and with growth and BW reported in this study were significantly different from zero. These results are consistent with genetic and phenotypic correlations reported in the literature (Arthur et al., 2001a, b; Crews et al. 2003a; Herd and Bishop, 2000; Robinson and Oddy, 2004; Schenkel et al., 2004). The genetic and phenotypic correlations between RFIp and RFIg were higher than 0.90, implying that they are almost the same trait. Hoque et al. (2005) reported the genetic and phenotypic correlation between RFIp and RFIg, respectively, to be 0.97 and 0.98 in Japanese black cattle. The genetic correlation between RFIg and RFIp observe in this study imply that approximately 15% of the variation in phenotypic residual feed intake was not captured by the genetic regression. Such variation may be purely environmental in nature and may account for the differences in heritability estimates obtained between RFIp and RFIg.

The correlations of DMI and FCR with ADG and metabolic BW are similar to published estimates as reviewed by Koots et al. (1994), as well as those reported by Liu et al., (2000), Arthur et al. (2001a, b), and recently by Schenkel et al. (2004) and Robinson and Oddy (2004). However, these correlations are in contrast with several earlier reports (Gill et al., 1986; Meissner et al., 1995, Gibb and McAllister, 1999), which indicated that the correlations between feed intake and gain or between intake and FCR are generally poor in feedlot cattle. Perhaps, recent developments in technological expertise for the estimation of feed consumption and animal performance using automated individual animal feeding systems (such as the radio frequency based GrowSafe® system used in this study) could be increasing our ability to accurately measure animal performance.

The high genetic correlations of DMI with ADG and MWT imply that a considerable proportion (76%) of the genetic variation in intake is associated with genetic differences in maintenance and growth, and the remainder represents only a small proportion of the total genetic variance. Nevertheless, the respective genetic SD of RFIp and RFIg (0.39, 0.67 kg DM/d) represent useful variation that probably reflect between animal differences in other metabolic processes not accounted for by differences in growth and BW (Crews et al., 2005; Herd et al., 2004). The strong phenotypic and genetic relationships of daily DMI with growth rate and body size imply that one-sided selection for faster growth rate and higher finish weights would lead to higher maintenance energy requirements and higher overall feed consumption, especially in mature breeding animals (Archer et al., 1999). Similarly, a one-sided selection against daily DMI may lead to reductions in growth and BW at maturity, which may be undesirable for the feedlot sector of the beef industry. The genetic and phenotypic relationships among ADG, DMI, and FCR obtained in this and other studies indicate that selection against FCR would reduce the amount of feed required for growth and thus be very beneficial to the feedlot operator. However, a strong correlation of FCR with growth raises questions in terms of its value to the improvement of overall production system efficiency as it may also lead to direct increases in mature BW, resulting in an increase in the cost of maintaining breeding herds (Barlow, 1984; Archer et al., 1999).

The only report in the literature comparing RFI to PEG in terms of genetic and phenotypic relationships with cattle performance is the study by Arthur et al. (2001a). The relationship of RFI or PEG with each other and with ADG, MWT and DMI obtained in the study by Arthur et al. (2001a) as well as in this study may indicate that selection for PEG or against RFI would be similarly beneficial in terms of the correlated reduction in feed intake with little effect (PEG) or no effect (RFI) on growth rate and no effect on body size. Arthur et al. (2001a) explain that indices of feed efficiency that incorporate linear combinations of measures of growth and metabolic body size seek to capture the variations among animals in energy utilization for growth and maintenance. This ensures that the cattle resulting from this form of selection would potentially be efficient both as feedlot animals and in the breeding herd. This is not the same with FCR, which is a ratio trait and selection to reduce FCR may not necessarily be correlated specifically to improvements in feed efficiency, but may only reflect selection for increased growth rate (Crews, 2005). The argument is that, the use of a ratio trait in selection may not translate into equivalent improvements in efficiency mainly because selection pressure may be disproportionately applied either to the numerator or the denominator (usually in favor of the component with the highest genetic variance).

High-RFIp steers generally had higher 23% higher FCR and consumed 22% more more feed compared to animals with low-RFIp, despite the lack of differences in ADG or metabolic BW. The associations also showed that the PEG above maintenance of high RFI animals was 24% compared to 29% in medium RFI and 34% in low RFI animals. High genetic and phenotypic correlations between RFI and PEG observed in this study are not surprising as both traits incorporate components of feed intake due to maintenance and to growth. Arthur et al. (2001a) reported strong genetic and phenotypic correlations between RFIp and PEG. These findings therefore indicate that responses to selection for PEG would be similar to the expected responses to selection against RFI. However, unlike RFI, PEG showed moderate to high genetic and phenotypic correlations with ADG in the present study indicating that, at least in some animals, higher PEG may be related to increased growth rate and subsequently BW.

This observation is in contrast to Arthur et al. (2001a) who observed that PEG was not related to rate of growth. This difference in findings between the present study and the study by Arthur et al. (2001a) may be related to the small differences in the methodology for computing expected feed intake for maintenance (required for computing PEG). This may be true since observations have shown that even RFI may not be phenotypically independent of growth and body size when expected feed intake is computed from feeding standards formulae, instead of from regression equations (Arthur et al., 2001b).

The relationships between RFI, DMI and FCR with measures of ultrasound and carcass merit obtained in this study generally agree with published estimates, except that the standard errors associated with the present estimates were rather high, making it difficult to judge whether the estimates were indeed different from zero. However, to the best of our knowledge, there is no report in the literature comparing PEG to RFI with respect to effects on carcass merit. Koots et al. (1994) reported a significant genetic correlation between FCR and lean meat yield (r = -0.32). Herd and Bishop (2000) reported significant phenotypic and genetic correlations between RFI and carcass lean percentage (r = -0.43 ± 0.23). In addition, Arthur et al. (2001b) reported weak and moderate phenotypic correlations between feed intake and backfat (r = 0.23) or LM area (r = 0.33), respectively. In the same study however, phenotypic correlations of RFI or FCR and ultrasound measures of backfat or rib eye area were not different from zero. A study by Richardson et al. (2001) showed that a single generation of selection against RFI was accompanied by a small reduction in body fat content.

Recently, Schenkel et al. (2004) reported a positive phenotypic and genetic correlations (r = 0.17, 0.16) between RFIp and backfat thickness, but there were no correlations between RFIp with intramuscular fat. The same authors found negative genetic correlations between FCR (r = -0.28) and RFIp (r = -0.17) with LM area. Robinson and Oddy (2004) reported genetic correlations of 0.48, 0.38, and 0.61 between rib fat (same as grade fat in the present study) and RFIp, FCR, and DMI, respectively. The corresponding respective genetic correlations with LM area were -0.24, 0.20, and 0.23. The same authors also reported genetic correlations of 0.22, 0.08 and 0.39 between RFIp, FCR, and DMI, and intramuscular fat, respectively. A serial slaughter study by Basarab et al. (2003) indicated that RFI computed from regression of ADG and MWT on intake showed weak but significant correlations with carcass fat (r = 0.14), carcass lean (r = -0.21), gain in backfat thickness (r = 0.22), gain in marbling score (r = 0.22) and empty body fat (r =

0.26). Differences in carcass merit, such as less marbling on efficient cattle, may not be considered a favorable response by the beef cattle industry.

Thus, evidence from this and other studies generally point towards a potential for small (5 ± 2%) reductions in carcass fatness and rate of gain in subcutaneous fat coupled with a slight improvement in carcass lean meat yield and yield grade (4-5%) following selection against RFI. However, the results on the differences in carcass merit between high, medium and low RFI groups indicate that, while low RFI is associated with an increased lean meat yield and yield grade, the animals have more than adequate backfat thickness and do not stand any risk of being downgraded for lack of external fat cover. In addition, differences in marbling score among the various groups were not significant. The observed phenotypic relationships of PEG with carcass and ultrasound merit in this study are comparable to the relationships of RFI with carcass merit.

The reported differences in carcass merit and body composition in this and other studies for animals differing in RFI may account for only a small proportion of the observed variations in energetic efficiency between these animals. It has therefore been suggested that other sources of variation such as differences in heat increment (especially associated with feeding and visceral metabolism), level of feeding activity and feeding behaviour, nutrient turnover, and digestive functions may account for part of the variation in RFI (Oddy and Herd, 2001; Richardson et al., 2001; Johnson et al., 2003). Further research efforts are therefore required to characterize the sources of variation between animals differing in RFI.

This study has indicated that, though RFI shows small but significant relationships with carcass fatness and leanness, the efficient animals had adequate carcass fatness and did not stand any risk of being downgraded for lack of external fatness. Phenotypic RFI was phenotypically, but not genetically independent of ADG and metabolic BW. However, though a weak phenotypic relationship was observed between genetic RFI and ADG, genetic RFI was genetically independent of ADG and metabolic BW. The relationships of carcass and ultrasound merit with PEG in beef cattle may be similar to the relationships with RFI. Animals with low RFI may show significant reductions in the energy requirement for maintenance and increase the PEG above maintenance. Partial efficiency of growth may be similarly robust (compared to RFI) as a measure of energetic efficiency, but its potential relationships with growth rate may be a disadvantage to overall production efficiency in mature animals.

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Table 3.1. Ingredients and composition of experimental diets

Diet ingredient (% as fed basis)	Year 1	Years 2 and 3
Dry-rolled corn	80.00	-
Barley grain	-	64.50
Oat grain	-	20.00
Alfalfa hay	13.50	9.00
Beef feedlot supplement[a]	5.00	5.00
Canola oil	1.50	1.50
DM, %	90.50	88.90
Composition, DM basis		
ME, Mcal/kg	2.90	2.91
CP, %	12.50	14.00
NDF, %	18.30	21.49
ADF, %	5.61	9.50

[a] Contained 440 mg/kg of monensin, 5.5% Ca, 0.28% P, 0.64% K, 1.98% Na, 0.15% S, 0.31% Mg, 16 mg/kg I, 28 mg/kg Fe, 1.6 mg/kg Se, 160 mg/kg Cu, 432 mg/kg Mn, 432 mg/kg Zn, 4.2 mg/kg Co, as well as a minimum of 80,000 IU /kg vitamin A, 8,000 IU/kg vitamin D, and 1,111 IU/kg Vitamin E.

Table 3.2. Number of records, overall mean, SD, heritability, and additive genetic variance for the traits considered in the present study

Traits	Number of Animals	Mean	SD	Heritability	Additive Variance
Phenotypic RFI, kg/d	464	0.00	0.88	0.21 ± 0.12	0.15
Genetic RFI, kg/d	464	-0.14	1.01	0.42 ± 0.15	0.45
Dry matter intake, kg/d	464	10.45	1.61	0.54 ± 0.15	1.34
Feed conversion, kg DM/kg gain	464	7.29	1.26	0.41 ± 0.15	0.64
Partial efficiency of growth	464	0.29	0.06	0.56 ± 0.16	0.0021
Average daily gain, kg/d	464	1.46	0.27	0.59 ± 0.17	0.04
Metabolic size, kg$^{0.75}$	464	90.22	10.47	0.31 ± 0.14	24.24
Final BW, kg	464	458.75	65.65	0.32 ± 0.14	1226.00
Ultrasound backfat, mm	464	9.33	3.51	0.59 ± 0.14	5.38
Ultrasound LM area, cm^2	464	83.37	10.62	0.39 ± 0.13	38.58
Ultrasound marbling score	464	5.21	0.79	0.75 ± 0.16	0.41
Harvest weight, kg	464	535.83	60.26	0.50 ± 0.15	1789.00
Carcass weight, kg	381	312.32	31.88	0.33 ± 0.14	352.90
Carcass grade fat , mm	381	10.83	4.31	0.51 ± 0.15	8.61
Carcass LM area, cm^2	381	83.94	9.19	0.45 ± 0.15	35.99
Carcass marbling score	381	2.51	0.53	0.49 ± 0.16	0.12
Carcass lean meat yield, %	381	57.83	3.81	0.63 ± 0.17	9.40
Carcass yield grade	381	1.73	0.72	0.58 ± 0.18	0.30
Carcass quality grade	381	2.49	0.66	0.30 ± 0.16	0.16

Table 3.3. LS means (± SE) for different measures of energetic efficiency, feed intake, growth, and BW of composite steers differing in phenotypic residual feed intake (RFIp).

Traits	Phenotypic residual feed intake group[a]			P value[b]
	High	Medium	Low	
Number of animals	139	183	142	
Phenotypic RFI, kg DM/d	0.98 ± 0.04^{d}	-0.02 ± 0.03^{e}	-0.95 ± 0.04^{f}	<0.001
Genetic RFI, kg DM/d	0.85 ± 0.05^{e}	-0.18 ± 0.03^{d}	-1.08 ± 0.04^{f}	<0.001
Feed conversion, kg DM/kg gain	$8.11^{d} \pm 0.13$	$7.17^{e} \pm 0.12$	$6.59^{f} \pm 0.13$	<0.001
Daily DMI, kg/d	$11.63^{d} \pm 0.14$	$10.56^{e} \pm 0.12$	$9.53^{f} \pm 0.13$	<0.001
Partial efficiency of growth	$0.24^{f} \pm 0.004$	$0.29^{e} \pm 0.003$	$0.34^{d} \pm 0.004$	<0.001
ME intake, kcal/(kg$^{0.75}$ · d)	$301.10^{d} \pm 1.98$	$277.92^{e} \pm 1.67$	$252.81^{f} \pm 1.80$	< 0.001
Metabolic BW, kg$^{0.75}$	90.53 ± 1.16	89.68 ± 1.03	90.39 ± 1.13	0.73
ADG	1.47 ± 0.03	1.49 ± 0.03	1.48 ± 0.03	0.66
Final BW, kg	459.9 ± 7.3	455.0 ± 6.7	461.7 ± 7.3	0.62
Harvest weight, kg	543.8 ± 6.7	542.9 ± 6.0	533.4 ± 6.5	0.22

[a] Residual feed intake groups are defined as High, RFI is > 0.5 SD above the mean; Medium, RFI is ± 0.5 SD above and below the mean; Low, RFI is < -0.5 SD below the mean.

[b] P value from overall F test.

[d, e, f] Within a row, means without a common superscript letter differ ($P < 0.05$).

Table 3.4. Least squares mean (± SE) for ultrasound and carcass merit of composite steers differing in phenotypic residual feed intake (RFI).

Trait	Phenotypic residual feed intake group[a] High	Medium	Low	*P* value[b]
Number of animals	*139*	*183*	*142*	
Gain in ultrasound backfat, mm/d	0.038^{d} ± 0.002	0.031^{e} ± 0.001	0.029^{e} ± 0.002	<0.001
Gain in ultrasound LM area, cm^2/d	0.167 ± 0.005	0.16 ± 0.004	0.169 ± 0.004	0.78
Gain in ultrasound marbling (x 10^{-2})	0.72 ± 0.04	0.69 ± 0.03	0.71 ± 0.04	0.78
Ultrasound backfat, mm	9.86^{d} ± 0.31	8.62^{d} ± 0.26	8.27^{e} ± 0.29	<0.001
Ultrasound LM area, cm^2	83.44 ± 0.80	83.47 ± 0.63	83.42 ± 0.72	0.98
Ultrasound marbling score	5.21 ± 0.08	5.06 ± 0.07	5.12 ± 0.08	0.21
Number of steers	*106*	*164*	*111*	
Carcass weight, kg	318.30 ± 5.70	313.85 ± 4.80	310.69 ± 4.33	0.19
Carcass LM area, cm^2	84.23 ± 0.98	84.39 ± 0.81	83.30 ±0.95	0.53
Carcass grade fat , mm	11.80^{d} ± 0.46	9.76^{e} ± 0.38	9.59^{e} ± 0.45	<0.001
Carcass marbling score	2.55 ± 0.06	2.45 ± 0.05	2.44 ± 0.06	0.23
Carcass lean meat yield, %	56.95^{e} ± 0.44	58.61^{d} ± 0.36	59.00^{d} ± 0.45	0.003
Carcass yield grade	1.84^{d} ± 0.08	1.65^{d} ± 0.06	1.52^{e} ± 0.08	0.02

[a] Residual feed intake groups are defined as High, RFI is > 0.5 SD above the mean; Medium, RFI is ± 0.5 SD above and below the mean; Low, RFI is < -0.5 SD below the mean.

[b] *P* values from overall F test.

[d, e, f] Within a row, means without a common superscript letter differ ($P < 0.05$).

Table 3.5. Phenotypic (below diagonal) and genetic (± SE) correlations (above diagonal) among different measures of feed efficiency and feed intake in composite steers.

Traits[a]	RFIp	RFIg	FCR	PEG	DMI
RFIp		0.92 ± 0.07	0.62 ± 0.09	-0.87 ± 0.06	0.73 ± 0.18
RFIg	0.97		0.78 ± 0.10	-0.94 ± 0.03	0.65 ± 0.16
FCR	0.52	0.67		-0.78 ± 0.10	0.28 ± 0.23
PEG	-0.83	-0.88	-0.68		-0.51 ± 0.17
DMI	0.64	0.52	0.30*	-0.30*	

[a] RFIp = phenotypic residual feed intake (kg DM/d); RFIg = genetic residual feed intake (kg DM/d); FCR = feed conversion ratio (kg DM/kg gain); PEG = partial efficiency of growth.DMI = daily dry matter intake (kg/d).

* Phenotypic correlation is significantly different from zero at $P < 0.01$ critical threshold; All other phenotypic correlations were significantly different from zero at the $P < 0.001$ threshold.

Table 3.6. Phenotypic and genetic relationships of different measures of energetic efficiency and feed intake with growth rate, BW and ultrasound traits of composite steers.

Traits[a]	ADG	MWT	UBF	UMAR	ULMA
Phenotypic correlations					
RFIp	0.0002	-0.007	0.25**	0.03	0.09
RFIg	-0.21**	-0.03	0.20*	-0.01	0.05
FCR	-0.69***	0.20**	-0.04	-0.09	-0.09
PEG	0.35***	0.17*	-0.21*	-0.04	-0.03
DMI	0.60***	0.65***	0.41***	0.20**	0.35***
Genetic correlations (± SE)					
RFIp	0.46 ± 0.45	0.27 ± 0.33	0.35 + 0.30	0.32 + 0.29	-0.52 + 0.32
RFIg	-0.04 ± 0.25	0.12 ± 0.30	-0.04 + 0.22	0.44 + 0.19	-0.65 + 0.20
FCR	-0.59 ± 0.16	0.06 ± 0.32	-0.29 + 0.21	0.08 + 0.23	0.54 + 0.23
PEG	0.55 ± 0.16	-0.21 ± 0.25	0.02 + 0.20	-0.56 + 0.16	-0.76 + 0.18
DMI	0.87 ± 0.09	0.87 ± 0.10	0.29 + 0.19	0.53 + 0.17	0.44 + 0.20

[a] RFIp = phenotypic residual feed intake (kg DM/d); RFIg = genetic residual feed intake (kg DM/d); FCR = feed conversion ratio (kg DM/kg gain); DMI = daily dry matter intake (kg/d); PEG = partial efficiency of growth; ADG = average daily gain (kg/d); HH = hip height (cm); UBF = ultrasound backfat (mm); UMAR = ultrasound marbling score; ULMA = ultrasound LM area (cm²).

† ($P < 0.10$); * ($P < 0.05$); ** ($P < 0.01$); *** ($P < 0.001$).

Table 3.7. Phenotypic and genetic correlations of different measures of energetic efficiency and feed intake with carcass merit of composite steers.

Traits[a]	CWT	CGF	CLMA	LMY	CMAR	CYG
Phenotypic correlations						
RFIp	0.26**	0.23**	0.09	-0.21**	0.17*	0.22**
RFIg	0.18*	0.19*	0.08	-0.16*	0.14*	0.17*
FCR	-0.06	0.02	0.02	0.01	0.0001	0.01
PEG	-0.03	-0.14*	-0.06	0.09	-0.07	-0.09
DMI	0.61***	0.40***	0.24*	-0.33***	0.29**	0.36***
Genetic correlations (± SE)						
RFIp	0.05 ± 0.38	0.33 ± 0.29	-0.64 ± 0.26	-0.54 ± 0.29	0.28 ± 0.38	0.03 ± 0.47
RFIp	-0.03 ± 0.30	0.27 ± 0.24	-0.69 ± 0.32	-0.43 ± 0.37	0.18 ± 0.26	0.09 ± 0.32
FCR	-0.28 ± 0.31	0.07 ± 0.26	0.28 ± 0.27	0.19 ± 0.25	-0.10 ± 0.27	-0.02 ± 0.27
PEG	-0.01 ± 0.29	-0.32 ± 0.22	-0.18 ± 0.25	0.12 ± 0.23	-0.11 ± 0.24	-0.24 ± 0.23
DMI	0.66 ± 0.17	0.49 ± 0.19	0.21 ± 0.13	-0.50 ± 0.18	0.50 ± 0.21	0.67 ± 0.16

[a] RFIp = phenotypic residual feed intake (kg DM/d); RFIg = genetic residual feed intake (kg DM/d); FCR = feed conversion ratio (kg DM/kg gain); PEG = partial efficiency of growth; DMI = daily dry matter intake (kg/d); CWT = carcass weight (kg/d); CGF = Carcass grade fat (mm); CLMA = carcass LM area (cm^2); LMY = lean meat yield(%); CMAR = carcass marbling score; CYG = carcass yield grade.

† ($P < 0.10$); * ($P < 0.05$); ** ($P < 0.01$); *** ($P < 0.001$).

CHAPTER 4

Relationships of Feedlot Feed Efficiency, Performance, and Feeding Behaviour with Metabolic Rate, Methane Production, and Energy Partitioning in Composite Cattle[2]

4.1 Introduction

The high cost of feeding in beef cattle production means that profitability depends on the efficient and productive utilization of feed for maintenance and growth, with minimal excesses and losses. There is considerable phenotypic and genetic variation in measures of beef cattle feed efficiency, such as feed conversion ratio (**FCR**), residual feed intake (**RFI**), and partial efficiency of growth (**PEG**; Archer et al., 1999; Arthur et al., 2001). Thus, improvements in feed efficiency would lead to cost reduction and better overall production efficiency. Residual feed intake is the difference between an animal's actual feed intake and its expected intake based on the BW and growth rate over a specified period.

Considerable research progress has been made in defining the variation in RFI among different biological types of cattle. However, the biological reasons underlying the observed variations are generally unknown, though several mechanisms have been proposed (Johnson et al., 2003; Richardson and Herd, 2004). According to these authors, the sources that may contribute to the variation in RFI may include feed intake, digestion of feed, heat increment, protein turnover and overall tissue metabolism, feeding behaviour and activity, body composition and rate of gain, BW, prolificacy, as well as several other presently unknown factors.

Techniques employed in nutritional energetics of ruminants have classically been concerned with the partitioning of dietary energy into faecal, urinary, methane, heat, and recovered or product energy. Potential between animal variations in the partitioning of dietary energy into various sources may account for part of the observed differences among animals in RFI. A study by Basarab et al. (2003) indicated considerable differences in the size of visceral organs that were associated with heat production and energy retention between animals differing in RFI. In a recent study, Richardson et al. (2004) reported significant metabolic differences in Angus steers divergently selected for RFI. Recent estimates (Herd et al., 2002; Okine et al., 2003) also indicate considerable differences in methane and manure production between animals differing in RFI.

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[2] A version of this chapter has been published, Nkrumah et al., 2006, J. Anim. Sci. 84: 145-153.

Generally, there are considerable variations among cattle in energy utilization and partitioning. These are mainly related to differences in dietary energy losses (faecal, methane, and urinary), heat production (**HP**), and energy retention (Delfino and Mathison, 1991; Saama and Mao, 1995; Basarab et al., 2003). The present study examined the relationships of feedlot feed efficiency, performance, and feeding behaviour with metabolic rate, digestion, and energy partitioning in beef cattle ranked by RFI.

4.2 Materials and Methods

4.2.1 Animals and Diets

Twenty-seven Composite steers sired by Angus or Charolais bulls were used in the study. Post-weaning feedlot performance and feed efficiency tests using the GrowSafe automated feeding system (GrowSafe Systems Ltd., Airdrie, Alberta, Canada) were carried out for a total of 306 animals over 2 yrs (two test groups per year with approximately 80 animals per test) at the University of Alberta's Kinsella Research Station. Details of the procedures for the feedlot tests are as given in chapter 3 and Nkrumah et al. (2004). At the end of each feedlot test, steers were ranked by their RFI and selected to be halter-trained for use in the metabolic and digestion trials at the University of Alberta's Metabolic Research Center, Edmonton. Standard deviations above and below the mean RFI were used to group the selected steers into high-RFI (RFI is > 0.5 SD above the mean, n = 11), medium-RFI (RFI is ± 0.5 SD above and below the mean, n = 8), and low-RFI (RFI is < -0.5 SD below the mean, n = 8). Respective BW ± SD for the high-RFI, medium-RFI, and low-RFI groups during the measurements were 495.6 ± 12.7 kg, 529.1 ± 18.6 kg, and 501.2 ± 15.5 kg.

The feedlot test diets for the 2 yrs are shown in Table 3.1. In each year, the same feedlot test diet was used in subsequent metabolic and digestion trials. Corn was used in yr 1, instead of barley and oat, because of a feed barley shortage that particular yr. However, the diets used in both years were formulated to contain similar levels of ME. At the Metabolic Research Center in Edmonton, animals were housed individually in adjacent holding pens in a climate-controlled thermoneutral environment and adapted to individual metabolism crates and confinement-type respiration calorimetry stanchions. Each experimental period consisted of a 14-d adaptation period during which steers were acclimated (or reacclimated if previously used in a trial), gentled, and gradually brought to the specific feeding level. All steers in the study were cared for according to the guidelines of the Canadian Council on Animal Care (CCAC, 1993).

4.2.2 Digestion Trial Procedure

Steers were individually fed in metabolic crates after acclimation and achievement of full feeding level [2.5 x estimated NRC, 1996 maintenance requirement (0.077 Mcal NEm/BW$^{0.75}$)]. Animals were weighed twice during the acclimation period and the average BW was used to determine the 2.5 times NEm feeding level. The metabolic crates permitted steers to lie down or stand during the trial. The digestibility trial consisted of a 5-d period during which a total collection of faeces and urine was made. Aliquots of feed, orts (feed refusals), faeces (10%), and pre-acidified urine (5%) were collected daily (after thorough mixing) and stored at –20°C for later processing and analyses. Feed, orts, and faecal samples were dried in a forced-air oven at 60°C for 72 h and ground in a Wiley mill (Arthur H. Thomas, Philadelphia, PA) to pass a 1-mm screen.

Dry matter contents of the feed, orts, and faeces were determined by oven drying at 100°C to a constant weight. A standard macro-Kjeldahl procedure (AOAC, 1980) was used to determine N in feed, orts, faeces, and urine samples. Gross energy contents of feed, faeces, orts, and urine were determined in an automatically controlled Parr adiabatic oxygen bomb calorimeter (Parr Instrument Co., Inc. Moline, IL). Neutral detergent fiber was determined according to the procedure of Van Soest et al. (1991). Acid detergent fiber was determined according to AOAC (1997). These analyses were determined in the ANKOM 200 fiber analyzer (Ankom Technology Corp., Fairport, NY). The NDF and ADF analyses were carried out in triplicate, with the intra-sample coefficient of variation for fiber determination less than 5% in 95% of samples.

4.2.3 Indirect Calorimetry Procedure

Oxygen consumption and methane production were measured in a four-chamber, open-circuit, indirect calorimetry system (Delfino et al., 1988). The calorimetry system is designed for individual animals to stand or lie down in stanchions, with their heads in hoods. The hoods were located in a climate-controlled thermoneutral environment and animals were randomly allocated to the calorimetry hoods after acclimation. The calorimetry system was designed such that air could be drawn from the hoods at a mean oxygen concentration of 20%. Respired air was passed through Drierite (W. A. Hammond Drierite Co. Ltd, Xenia, OH) to remove water vapor before passing through a paramagnetic oxygen analyzer (Servomex Inc., Sussex, UK) or methane analyzer (Model 880A Infrared Analyzer, Rosemount Analytical, Orville, OH). A Foxboro 823 IFO integral flow orifice with cell transmitter (Invensys Systems, Inc., Foxboro, MA) was used to measure airflow rate. Pressure was measured with a Foxboro 821AL absolute pressure transmitter (Invensys Systems, Inc., Foxboro, MA). Temperature and relative humidity were also measured by cellular temperature and relative humidity transmitters (General Eastern, Fairfield, CT). The system also allowed measurement of the concentration of ambient oxygen.

The calorimetry system was calibrated by the N injection method (by releasing a weighed amount of N gas into the system) as described by Young et al. (1984). Two 16-h measurements at 3-d intervals were obtained from each steer at the estimated (NRC, 1996) 2.5 times maintenance requirement feeding level after the digestion trial. In order to estimate heat increment of feeding (**HIF**), oxygen consumption was also measured at 1.2 times maintenance feeding level. Steers were kept on the 1.2 times maintenance feeding level for 5 d before measurements were made in order to remove residual feed due to the higher feeding level from the gut.

4.2.4 Calculations and Statistical Analyses

Procedures for obtaining the measures of feedlot performance and feed efficiency have been described previously in chapter 3. Briefly, each animal's ADG during the feedlot test was computed as the coefficient of the linear regression of BW (kg) on time (d) using the regression procedure of SAS (SAS Institute, Inc., Cary, NC, version 9.1). The metabolic BW of each animal over the feedlot test period was computed as the mid-point $BW^{0.75}$ of a 70-d test. The total feed intake of each animal over a 70-d test period was used to compute the daily DMI. Residual feed intake was computed for each animal as the difference between actual DMI and predicted expected daily DMI based on the ADG and metabolic BW over the test period using procedures described by Arthur et al. (2001). The partial efficiency of growth (i.e., energetic efficiency for ADG) above maintenance of each animal was computed as the ratio of ADG to the difference between average daily DMI and expected DMI for maintenance (**DMIm**) (Arthur et al., 2001), where DMIm was computed according to NRC (1996). Feed conversion ratio was computed as the ratio of DMI to ADG on test.

The feedlot behaviour traits studied were daily feeding frequency (**FF**) and daily feeding duration (**FD**). Procedures for determining feeding behaviour from the GrowSafe System have previously been described (Basarab et al., 2003). Daily feeding frequency in this study was defined as the number of independent feeding events for a particular animal in a day. A feeding event starts when an animal's transponder is first detected and ends when the time between the last two transponder readings was greater than 300 seconds or when a different transponder number is encountered. Daily feeding duration was computed as the sum of the difference between feeding event end-times and start-times per day for each animal. It was equal to the total number of minutes each day spent in feeding-related activities (prehension, chewing, backing away from the bunk and chewing, socializing, scratching, or licking) at the feedbunk.

All energy intake and partitioning values during the post-feedlot trial were expressed per unit of metabolic BW (i.e., $BW^{0.75}$) and have been adjusted to 24-h basis. With the exception of HIF, which by convention, must be estimated using two different feeding levels, all energy partitioning and digestibility values reported in the study are measurements taken at the 2.5 times maintenance feeding level. Digestible energy was calculated by multiplying the daily intake energy by the energy digestibility of the diet from each animal. The energy lost as methane was calculated as the total methane produced in liters per day at standard temperature and pressure (**STP**) x 9.45 kcal/L (Brouwer, 1965). Metabolizable energy (kcal/$kg^{0.75}$) was calculated by subtracting energy losses (kcal/$kg^{0.75}$) in urine and methane from DE (kcal/kg$^{0.75}$). Heat production (kcal/kg$^{0.75}$) was computed as (–4.90 kcal/L O_2) x (volume of expired air at STP) x (oxygen in exhaust air - oxygen in inlet air at STP). This approach has been shown to give accurate estimates of HP (± 1.2%) (McLean and Tobin, 1990). Heat increment of feeding was calculated as the change in HP per unit change in ME intake for the same animal (McDonald et al., 2002). Retained energy (RE) (kcal/kg$^{0.75}$) was calculated as the difference between ME and HP (NRC, 1996) at 2.5 times maintenance requirement.

Data were analyzed using a SAS mixed model (SAS Institute, Inc., Cary, NC, version 9.1), which included fixed effects of RFI group (high, medium, and low), year (one and two), test group within year (two groups per year), and random effects of metabolic crate or calorimetry chamber, and sire of animal. All interaction terms that were not significant for a trait ($P > 0.10$) were dropped from the final model. There was no RFI group-by-year interaction on any of the traits considered. Mean separation among RFI groups for different test traits was carried out by least-squares using the PDIFF option in SAS. The PROC CORR of SAS was used to obtain Pearson partial phenotypic correlations adjusted for the linear effects of DMI and the fixed effect of year.

4.3 Results

There were differences among the different groups of RFI steers selected for the study in PEG ($P < 0.001$), FCR ($P = 0.01$), DMI ($P = 0.01$), and the two measures of feeding behaviour ($P < 0.01$), but not in metabolic BW or ADG ($P > 0.10$; Table 4.1). These differences provided adequate variation among the animals for determining the relationships of the different measures of feed efficiency, performance, and behaviour with the measures of metabolic rate, digestion, and energy partitioning considered in the study. Daily faecal DM, methane, orts, urine, urinary N excretion, and apparent digestibility of dietary components are presented in Table 4.2. Table 4.3 shows the associations between RFI and measures of daily energy partitioning. In Table 4.4, the phenotypic correlations between the feedlot measures of performance, efficiency, and feeding behaviour with daily energy partitioning have been presented.

Daily DMI of the steers at the 2.5 times feeding level averaged 80.02 ± 7.78 g/kg$^{0.75}$, and this corresponded to an average GE intake of 375.2 ± 39.5 kcal/kg$^{0.75}$. There were no differences in daily DMI or GE intake between the different RFI groups during the post-feedlot trial ($P > 0.10$). Of the mean daily GE intake of the steers, 24.98 ± 8.39% was recovered in the faeces, and 3.89 ± 1.22 and 2.67 ± 0.85 were recovered as methane gas and in the urine, respectively. Thus, the mean DE and ME in the study were approximately 75% and 68%, respectively, with a corresponding ME to DE ratio of 0.91. Daily HP averaged 149.0 ± 19.72 kcal/kg$^{0.75}$ and formed approximately 59% of the average daily ME intake, with a corresponding ME retention efficiency of 39%. Feedlot RFI was correlated with daily methane production ($P < 0.05$) and was about 28% and 24% lower in low-RFI animals compared to high or medium-RFI animals, respectively. These differences were generally consistent over the entire 16 h calorimetry period. Residual feed intake was also negatively correlated with daily DE and ME ($P < 0.05$). High-RFI steers recovered 9.7% lower DE and 10.2% lower ME from their daily feed consumed compared to low-RFI steers.

Daily HP and energy retention were highly significantly associated with feedlot residual feed intake ($P < 0.001$). Heat production was 21% and 10% lower in low-RFI steers compared to steers with high or medium-RFI, respectively. Consistent with this is the lower RE (44% and 23%) in high and medium-RFI steers, respectively, compared to low-RFI steers. Simple regression analyses showed that differences in feedlot DMI, post-feedlot HP, and post-feedlot ME (kcal/d), respectively, accounted for 20%, 48%, and 16% of the variation in feedlot RFI among the animals. The regression equation can be represented as feedlot RFI = -6.52 + 0.320 x DMI (kg/d; feedlot) + 0.031 x HP (kcal/kg$^{0.75}$; post-feedlot) - 0.005 x ME (kcal/ kg$^{0.75}$; post feedlot). The differences in metabolizability were attributable mainly to the observed differences in DE (kcal/d) and methane production.

No significant differences were observed among the RFI groups in HIF, measured at two different feeding levels above maintenance ($P > 0.20$), though low-RFI steers had 32.6% lower HIF. There was a tendency for a negative association between RFI and digestibility of dietary CP ($r = -0.34$; $P < 0.10$) and DM ($r = -0.33$; $P < 0.10$). Daily faecal DM production was 15.5% and 8.1% higher in high and medium-RFI steers, respectively, compared to low-RFI steers, though these differences were not statistically significant ($P > 0.10$). The results for NDF and ADF indicated that NDF digestibility was generally lower in high-RFI compared to low-RFI steers, though differences were not statistically significant. Correlations of NDF and ADF digestibility with RFI were not different from zero ($P > 0.10$). Urinary N excretion was 17% higher in the urine of high-RFI steers compared to low-RFI steers, though these differences, as well as that of daily urine production, were not significant ($P > 0.10$).

The relationship of PEG with the various test traits consistently followed those with RFI. There were significant correlations ($P < 0.01$) between PEG with DE, ME, HP, RE, and methane production. Similarly, the PEG of the steers tended to be related to faecal output and digestibility of dietary components ($P < 0.10$). With the exception of daily heat production ($P < 0.05$), feedlot FCR of the steers was generally unrelated to any of the metabolic rate and energy partitioning traits considered in the study. Feedlot DMI showed positive associations with methane production ($P < 0.05$) and energy lost through urine ($P < 0.01$), but was negatively correlated with daily DE ($P < 0.05$), ME ($P < 0.01$), and RE ($P < 0.01$). There was also a tendency for feedlot DMI to affect faecal DM production (positive association), HP, and DM digestibility (negative association) ($P < 0.10$).

Daily feeding frequency of steers was positively related to HP and negatively related to NDF and ADF digestibility ($P < 0.05$), but was unrelated to other traits considered in the study ($P > 0.10$). Daily feeding duration showed significant correlations with faecal DM and methane production ($P < 0.01$) (positive associations), daily DE, ME, RE, and apparent digestibility of CP and DM ($P < 0.01$) (negative associations). Feedlot ADG was generally unrelated to the traits considered in the study, except a weak trend towards an association with daily DE and ME ($P = 0.12$). With the exception of daily urinary energy ($P < 0.05$), heat production, NDF, and ADF digestibility ($P < 0.10$), feedlot metabolic BW was generally not related to the traits considered in the study. The use of either corn or barley and oats in yr 1 or 2 did not result in any interactions between RFI and year in the analyses ($P > 0.10$). However, energy lost as methane (% of GE intake) was lower ($P < 0.01$) for the diet in yr 1 (corn-based diet; 3.25 ± 0.23 %) compared to the yr 2 diet (barley-based diet; 4.59 ± 0.30 %). Additionally, dietary and faecal NDF and ADF levels were lower ($P < 0.05$) for the corn-based diet compared to the barley-based diet.

4.4 Discussion

The identification of the metabolic and physiological reasons underlying the variation in feed efficiency in cattle that are similar in body weight and growth is a well-recognized pre-requisite for the effective planning of breeding strategies to select animals that are more efficient. In the present study, we considered several potential metabolic and physiological pathways that may influence feed efficiency. These include those pathways that are generally related to variations in the efficiency of conversion of GE into ME (due to differences in digestibility, generation of gases during rumen fermentation, absorption of nutrients, waste excretion, and heat production) and the subsequent efficiency of ME conversion to RE for maintenance and growth. The relationship of feedlot RFI with metabolic BW, ADG, DMI, FCR, and PEG of the animals selected for the post-feedlot study were consistent with those reported in the literature (Arthur et al., 2001; Basarab et al., 2003).

The present study identified significant differences in methane emission among animals differing in RFI, which represents the first experimental evidence demonstrating associations between RFI and methane production. Previous evidence (Herd et al., 2002; Okine et al., 2003) relating RFI to methane production were based only on estimates derived from the relationship between RFI and DMI, and resulted in about 5% difference between low-RFI and high-RFI animals in methane production. Data from the present study indicated that methane production was 25% lower in low-RFI animals compared to high animals, even though feed intake was similar during the measurement period. These differences correspond to approximately 16,100 L/annum less methane in low-RFI animals compared to high-RFI animals.

The mechanisms behind the observed differences among animals in methane emission, independent of intake, are unknown, but may be related to differences in metabolizability as well as possible individual animal differences in methane production. Methane production has been shown to be heritable ($h^2 = 0.42$) in humans (Flatz et al., 1985). According to Hackstein et al. (1996), there is a genetic link between methanogens and their hosts such that the presence of methanogenic bacteria in an animal requires a quality of the host that is under phylogenetic rather than dietary constraint. Whether this link has any effects on the type of methanogens that are dominant in the rumen of individual animals is unknown.

However, any inherent differences in animals that may lead to ecological changes in the rumen microbial eco-system may translate into differences in methane production. Increased methane production from high-RFI animals not only represents significant reductions in energetic efficiency but also has implications for the environmental sustainability of beef cattle production due to the significant contributions to atmospheric methane emissions. Agriculture in Canada contributes about 10% of the total Canadian green house gas emissions (Environment Canada, 2004), of which 2.6% is methane. The present study also identified a tendency towards differences in daily faecal DM production per unit of DMI, an observation consistent with previous estimates by Okine et al. (2003).

The study also indicated a trend towards associations between RFI and apparent digestibilities of dietary DM and CP. These differences in apparent digestibilities between RFI groups reported in the present study were consistent with Richardson et al. (1996) who reported higher DM digestibility in low-RFI steers than in high-RFI steers, and concluded that small differences in digestibility can result in large differences in feed efficiency. The differences in apparent digestibility observed in the present study between high-RFI and low-RFI animals were however weak (approximately 5%). A recent study by Channon et al. (2004) demonstrated significant genetic and phenotypic associations between RFI and traits that are indicative of the extent of starch digestion in the gastrointestinal tract of cattle. Russell and Gahr (2000) indicated that individual animal variations in factors such as the mechanism of digestion and absorption, rumen retention time and feeding behaviour may contribute to the variations between individual animals in diet digestibility.

Variation in rumen retention time among animals have been reported in cattle (Ørskov et al., 1988) and may be associated with differences in DMI or feeding behaviour. Significant differences in feedlot DMI among animals differing in RFI have been demonstrated in several studies (Arthur et al., 2001; Basarab et al., 2003; Nkrumah et al., 2004). In addition, considerable differences in feeding duration were observed among the animals in the different RFI groups in the present study. The differences in feeding duration in the present study were associated positively with differences in faecal and methane production, and negatively with DM and CP digestibility. These associations also translated into differences in daily DE and ME among the steers differing in RFI.

Significant differences in DE, ME, and RE among the animals of the different RFI groups were also demonstrated in the present study. Part of the variation in efficiency of energy retention has been attributed, among other factors, to a depression of metabolizability of the diet and the increase in the HIF at high levels of intake above maintenance (Ferrell and Jenkins, 1998). The results of the present study are generally in agreement with the above suggestions as the differences in feedlot DMI were also associated with significant reductions in DE, ME, and RE. However, to evaluate whether differences in daily methane production, DE, ME, or RE were due to inherent differences in the different RFI steers independent of the level of intake-associated effects, we re-run the data analysis with feedlot DMI included as a linear covariate in the statistical models of analyses. This did not eliminate the relationships of the above traits with RFI, demonstrating that part of the variation in the different RFI steers in DE, ME, and RE may indeed be independent of the level of intake.

The results of the present study therefore indicate that the higher DMI for animals with high-RFI may be partly related to the low metabolizability of consumed feed and the accompanying increased need to attain the level of energy intake required for maintaining BW and supporting body protein and fat accretion. According to the present results, the lowered metabolizability of feed in high-RFI steers in itself may be attributable, at least in part, to the reduced digestibility and increased faecal and methane production, but is less related to energy lost through urine. The present study however, failed to demonstrate significant differences among RFI groups in HIF above maintenance, partly due to the high within-RFI group variation in heat increment of feeding.

The mean of the ratio of ME to DE observed in the present study (0.91) is indeed higher than the 0.82 suggested by the beef cattle NRC (1996). Similar values have been reported for other studies (Rikhardsson et al., 1991). For feedlot steers on high grain diets that may also contain vegetable fats or ionophores such as monensin, the proportion of intake energy lost through urine and methane are considerably lower than for high roughage diets (Van der Honing and Steg, 1990). The practice of adding vegetable oils and ionophores to high grain feedlot diets, to reduce extreme cases of bloat, have especially been shown to depress rumen methanogenesis considerably (Mathison, 1997; McGinn et al., 2004), and may have contributed to the high ME to DE ratio in the present study.

The considerably higher heat production in high-RFI steers compared to low-RFI steers observed in the present study, despite the lack of differences in GE intake, may be attributable to differences among the animals in metabolic efficiency. Variation in energy expenditure related to differences in the size of visceral organs for instance, has been proposed as contributing significantly to the differences in heat production between animals with different RFI (Basarab et al., 2003). Residual feed intake is positively correlated with DMI and it has been demonstrated that increased DMI in cattle is generally accompanied by significant increases in the size of visceral organs (Ferrell and Jenkins, 1998). Indeed, a study by Basarab et al. (2003), which indicated a higher heat production from high RFI animals compared to low RFI animals, also reported significantly higher visceral organ weights in high-RFI steers compared to low-RFI steers.

According to Reynolds (2002), differences in visceral organ size contribute significantly to the variation in total oxygen consumption, and thus heat production, accounting for 40 to 50% of daily heat production. Additionally, Webster (1980) indicated that there is a strong linear relationship between protein synthesis and heat production and that marked differences in metabolic rate could almost entirely be explained by differences in protein synthesis. Not surprisingly, the greatest proportion of the protein synthesis and associated heat production takes place in visceral tissues such as the gastrointestinal tract and the liver, which are not normally associated directly with growth and meat production (Webster, 1980; Reynolds, 2002). An evaluation of the differences in expression of genes involved in protein turnover and associated heat production in certain metabolically active tissues, such as the liver and gastrointestinal tract, may help to explain part of the molecular mechanisms leading to variations in energy expenditure in cattle with similar BW and ADG. This may be even more important in ruminants due to the comparatively large size of the viscera in relation to the whole body.

This study has provided experimental evidence indicating significant associations between feedlot residual feed intake with methane production and measures of metabolic rate and dietary energy partitioning in beef cattle. The results show that differences in metabolizability (mainly digestibility and methane production), heat production, and energy retention are responsible for a major part of the variation between animals in residual feed intake. These findings should provide a basis for further research to better characterize the biological sources of variation in energetic efficiency in beef cattle. This will be useful for the efficient planning of breeding strategies to select animals that eat considerably less for a similar growth rate and body weight.

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Table 4.1. Relationship of residual feed intake with measures of feedlot performance, efficiency, and feeding behaviour of steers used in the study (LS means ± SE)

	Residual feed intake group[a]			
Traits	High	Medium	Low	*P* value[e]
Number of steers	*11*	*8*	*8*	
Residual feed intake, kg/d	1.25 ± 0.13[b]	-0.08 ± 0.17[c]	-1.18 ± 0.16[d]	<0.001
Feed conversion ratio, kg DM/kg gain	7.98 ± 0.23[b]	7.04 ± 0.29[c]	6.53 ± 0.30[d]	0.01
Partial efficiency of growth	0.26 ± 0.01[b]	0.31 ± 0.01[c]	0.38 ± 0.01[d]	<0.001
DMI, kg/d	11.62 ± 0.30[b]	11.07 ± 0.39[c]	9.62 ± 0.36[d]	0.01
Metabolic BW, kg$^{0.75}$	89.04 ± 2.57	92.21 ± 2.77	93.75 ± 2.87	0.48
ADG, kg/d	1.46 ± 0.20	1.51 ± 0.16	1.48 ± 0.16	0.39
Feeding duration, min/d	73.95 ± 4.34[b]	65.03 ± 4.69[c]	47.76 ± 4.85[d]	0.006
Feeding frequency, events/d	35.58 ± 3.01[b]	29.68 ± 3.38[c]	18.07 ± 3.49[d]	0.01

[a] Residual feed intake groups are defined as High, RFI is > 0.5 SD above the mean; Medium, RFI is ± 0.5 SD above and below the mean; Low, RFI is < -0.5 SD below the mean.

[b, c, d] Within a row, means without a common superscript letter differ ($P < 0.05$).

[e] *P* values from overall F test.

Table 4.2. Relationship of feedlot residual feed intake with faecal DM, urine and methane production, and digestion in beef cattle fed at 2.5 x their estimated (NRC, 1996) maintenance requirements (LS means ± SE)

Traits	Residual feed intake group[a]			*P* value[e]
	High	Medium	Low	
Number of steers	*11*	*8*	*8*	
Metabolic BW, $kg^{0.75}$	105.01 ± 2.00	110.22 ± 2.94	105.75 ± 2.45	0.32
DMI, $g/kg^{0.75}$	82.66 ± 2.03	78.77 ± 2.62	81.75 ± 2.69	0.35
Orts, % of DM offered	10.31 ± 2.11	17.77 ± 3.16	14.04 ± 3.21	0.14
Faecal DM production, g/kg DMI	272.13 ± 13.0	249.72 ± 16.79	234.22 ± 17.16	0.24
Methane, $L/kg^{0.75}$	1.71 ± 0.11^{b}	1.68 ± 0.14^{b}	1.28 ± 0.14^{c}	0.04
Urine production, $g/kg^{0.75}$	56.27 ± 4.62	49.62 ± 5.61	45.49 ± 5.47	0.25
Urine N, g/kg DMI	8.60 ± 0.60	8.92 ± 0.72	7.13 ± 0.74	0.19
Apparent digestibility, %				
DM	70.87 ± 1.97^{c}	73.40 ± 2.12^{bc}	75.33 ± 2.10^{b}	0.10
CP	69.76 ± 2.17^{c}	73.52 ± 2.32^{bc}	74.70 ± 2.29^{b}	0.09
NDF	17.29 ± 8.24	28.25 ± 8.51	31.49 ± 8.31	0.19
ADF	3.26 ± 8.94	10.07 ± 9.24	14.67 ± 9.03	0.43

[a] Residual feed intake groups are defined as High, RFI is > 0.5 SD above the mean; Medium, RFI is ± 0.5 SD above and below the mean; Low, RFI is < -0.5 SD below the mean.

[b, c, d] Within a row, means without a common superscript letter differ ($P < 0.05$).

[e] *P* value from overall F test.

Table 4.3. Relationship of feedlot residual feed intake with post-feedlot daily dietary energy flows in beef cattle fed at 2.5 x their estimated (NRC, 1996) maintenance requirements (LS means ± SE)

	Residual feed intake group^{a}			
Traits	High	Medium	Low	*P* value^{e}
Number of steers	11	8	8	
Intake energy, kcal/kg$^{0.75}$	384.77 ± 7.90	382.24 ± 6.26	387.98 ± 6.03	0.39
Faecal energy, kcal/kg$^{0.75}$	104.41 ± 4.89	96.02 ± 6.44	88.22 ± 6.65	0.16
Digestible energy, kcal/kg$^{0.75}$	265.18 ± 5.20^{c}	288.11 ± 7.07^{b}	293.78 ± 6.84^{b}	0.05
Methane energy, kcal/ kg$^{0.75}$	16.08 ± 1.01^{b}	15.90 ± 1.30^{bc}	12.09 ± 1.28^{c}	0.04
Urinary energy, kcal/kg$^{0.75}$	10.88 ± 0.64	9.36 ± 0.84	10.00 ± 0.81	0.35
Metabolizable energy, kcal/ kg$^{0.75}$	238.54 ± 5.41^{d}	248.73 ± 7.13^{c}	265.73 ± 7.36^{b}	0.02
Heat production, kcal/kg$^{0.75}$	163.97 ± 4.17^{b}	143.00 ± 5.54^{c}	129.32 ± 5.46^{d}	<0.001
Retained energy, kcal/kg$^{0.75}$	75.34 ± 7.22^{d}	104.30 ± 9.51^{c}	135.23 ± 9.82^{b}	<0.001
Heat increment, kcal/kcal ME	53.60 ± 10.54	53.18 ± 13.88	36.08 ± 14.35	0.58
Energy losses, (% GE intake)				
Methane	4.28 ± 0.26^{b}	4.25 ± 0.35^{b}	3.19 ± 0.34^{c}	0.04
Urine	2.55 ± 0.28	2.32 ± 0.30	2.47 ± 0.28	0.74
Faeces	28.80 ± 1.77	26.39 ± 2.01	24.18 ± 2.02	0.14
ME/DE	0.90 ± 0.01	0.91 ± 0.01	0.92 ± 0.01	0.16

[a] Residual feed intake groups are defined as High, RFI is > 0.5 SD above the mean; Medium, RFI is ± 0.5 SD above and below the mean; Low, RFI is < -0.5 SD below the mean. [b, c, d] Within a row, means without a common superscript letter differ (P < 0.05).

[e] *P* value from overall F test.

Table 4.4. Relationship of feedlot growth, feed intake, feed efficiency, and feeding behaviour with measures of post-feedlot digestibility and energy partitioning of steers[a]

Traits[b]	DFO	DMP	DDE	DME	DHP	DRE	DMD	CPD	NDF	ADF
RFI	0.33†	0.44*	-0.41*	-0.44*	0.68***	-0.67***	-0.33†	-0.34†	-0.005	0.11
FCR	-0.02	0.19	-0.06	-0.09	0.37*	-0.24	0.02	-0.09	0.04	0.07
PEG	-0.35†	-0.55**	0.45*	0.49**	-0.50**	0.62***	0.35†	0.32†	-0.20	-0.28†
DMI	0.32†	0.38*	-0.46**	-0.48**	0.31†	-0.53**	-0.32†	-0.16	0.13	0.20
ADG	0.23	0.05	-0.28‡	-0.27†	-0.09	-0.18	-0.23	-0.04	0.04	0.06
MBW	0.005	-0.03	-0.08	-0.10	-0.36†	0.07	-0.005	0.19	0.32†	0.30†
FF	-0.23	-0.14	0.22	0.22	0.42*	0.004	0.25	0.06	-0.48**	-0.46**
FD	0.54**	0.51**	-0.52**	-0.55**	0.25	-0.60**	-0.55**	-0.47*	0.23	0.36†

[a] Traits in the columns were measured during the feedlot trial and those on the rows during the post-feedlot animal house experiment.

[b] DFO = daily faecal DM output; DMP = daily methane production; DDE = daily DE; DME = daily ME; DHP = daily heat production; DRE = daily RE; DMD = apparent DM digestibility; CPD = apparent CP digestibility; NDF = neutral detergent fiber digestibility; ADF = acid detergent fiber digestibility; RFI = residual feed intake; FCR = feed conversion ratio; PEG = partial efficiency of growth; DMI = dry matter intake; MBW = metabolic BW; FF = daily feeding frequency; FD = daily feeding duration.

† ($P < 0.10$); * ($P < 0.05$); ** ($P < 0.01$); *** ($P < 0.001$).

CHAPTER 5

Genetic and Phenotypic Relationships of Feeding Behaviour and Temperament with Growth, Feed Intake, Feed Efficiency, Ultrasound, and Carcass Merit of Composite Cattle.

5.1 Introduction

There are ongoing worldwide research efforts directed towards genetic improvements in overall beef cattle production system efficiency in order to reduce production cost and improve profitability. Genetic parameter estimates on economically relevant traits (**ERT**) provide the basic information required to develop selection strategies and to predict expected rates of direct and correlated responses to selection. Data on traits such as feeding behaviour and temperament may be incorporated into genetic evaluations either as indicator traits that show correlations with certain ERT or because the behaviour traits may have a direct economic value. Despite the potential effects of selection for certain ERT on animal behaviour (Schwartzkopf-Genswein et al., 2003), there is a paucity of information on the relationships of measures of behaviour with performance, feed efficiency, and carcass merit of farm animals.

The feeding behaviour of individual animals is generally consistent and highly repeatable over a specified period (Gibb et al., 1998), and may potentially be used to predict differences in animal performance and efficiency (Oddy and Herd, 2001). Several efforts have been made in the past to document the feeding behaviour of individual animals and determine how behaviour differences relate to traits such as health, growth, feed intake, and feed efficiency (Keys et al., 1978; Sowell et al. 1998; Schwartzkopf-Genswein et al., 2003; Cammack et al., 2005; Robinson and Oddy, 2004). Despite all these efforts, very little is still known about the specific relationships (especially genetic relationships) of feeding behaviour with many ERT in beef cattle. Robinson and Oddy (2004) reported that daily feeding time and eating sessions of tropically adapted and temperate steers and heifers were moderately heritable and showed genetic and phenotypic relationships with performance, feed efficiency, and carcass merit. Recently, Cammack et al. (2005) also reported that measures of feeding behaviour in ram lambs were moderately heritable, and showed genetic and phenotypic correlations with feed intake and measures of feed efficiency.

The temperament of an animal may be defined as its behavioural response to handling by humans (Burrow and Corbet, 2000). Earlier measures of temperament can be categorized into restrained methods such as the subjective 'chute score' and non-restrained methods such as those based on visual observations in a pen (pen score). Burrow et al. (1988) developed flight speed (based on the measurement of the time to cover a fixed distance while exiting a confined area) as an objective measure of temperament in non-restrained situations. Burrow and Corbet (2000) reported the heritability of flight speed in Zebu or Zebu-derived cattle to range from moderate to high. A number of studies have also been conducted to determine the relationships of FS with growth rate, carcass merit, meat quality (Fox et al., 2004; Burrow and Dillon, 1997; Voisinet et al., 1997), and feed efficiency (Fox et al., 2004). Results from these studies indicated that animals with excitable temperaments have lower growth rate, BW and meat tenderness, but no phenotypic relationships were found with feed efficiency. The objective of this study was to provide genetic parameter estimates for measures of feeding behaviour and temperament, and to determine their genetic and phenotypic relationships with growth, BW, feed intake, feed efficiency and carcass merit of beef cattle.

5.2 Materials and Methods

5.2.1 Animals and Management

Growth, feed intake, feeding behaviour, temperament, ultrasound and carcass data were collected on composite steers sired by Angus, Charolais or University of Alberta Hybrid bulls between 2002 and 2005. The dams used were produced from crosses among three composite cattle lines, namely Beef Synthetic 1 (BS1), Beef Synthetic 2 (BS2) and Dairy x Beef Synthetic (DBS) (Goonewardene et al., 2003). Details of the procedures for the feedlot tests were given in Chapter 3 (Nkrumah et al., 2004). Briefly, the animals were managed and tested under feedlot conditions using the GrowSafe automated feeding system (GrowSafe Systems Ltd., Airdrie, Alberta, Canada) at the University of Alberta's Kinsella Research Station. The animals weighed 353.0 (SD = 61.3) kg and were 252 (SD = 42) days of age at the beginning of testing. Two tests made up of approximately 80 animals per test were conducted each year. The animals used in the study were cared for according to the guidelines of the Canadian Council on Animal Care (CCAC, 1993).

Yr 1 steers were fed free choice a backgrounding diet of mainly alfalfa-brome hay with oats supplemented with corn grain and feedlot mineral supplement to promote a growth rate of just under 1.0 kg/d for approximately 30 d. This period was followed by a 30-d pre-test adjustment period in which the amount of corn in the backgrounding diet was adjusted up gradually to introduce the animals to the test diet and the feeding system. This was done to allow them to adapt to the diet and learn to feed from the test facility. The test diet in yr 1 was composed of 80.0% dry-rolled corn, 13.5% alfalfa hay pellet, 5% feedlot supplement (32% CP beef supplement) and 1.5% canola oil, supplying approximately 2.90 Mcal/kg of ME and 12.5% CP. In yr 2 and 3, the same test procedures were used, but the test diet contained 64.5% barley grain, 20% oat grain, 9.0% alfalfa hay pellet, 5.0% beef feedlot supplement and 1.5% canola oil, supplying 14.0% CP and 2.91 Mcal/kg of ME. Corn was used in yr 1, instead of barley and oat, because of a feed barley shortage that particular year.

5.2.2 Traits, Definitions and their Derivations

Procedures for obtaining the measures of feedlot performance and feed efficiency have been described previously in Chapter 3 (Nkrumah et al., 2004). Linear regression in SAS (SAS Institute, Inc., Cary, NC, version 9.1.3) of weekly or fortnightly BW measurements against time (d) was used to derive ADG, final BW and mid-test metabolic size (**MWT**, $BW^{0.75}$) for each steer. The total feed intake of each animal over a 70-d test period was used to compute the daily DMI. Feed conversion ratio (**FCR)** was computed as the ratio of DMI to ADG on test. The partial efficiency of growth (PEG; i.e., energetic efficiency for ADG above maintenance) of each animal was computed as the ratio of ADG to the difference between average daily DMI and expected DMI for maintenance (**DMIm**) (Arthur et al., 2001), where DMIm was computed according to NRC (1996).

Residual feed intake (**RFI**, an alternative measure of feed efficiency) was calculated both from phenotypic regression (**RFIp,** Arthur et al., 2001) or genetic regression (**RFIg**, Hoque and Oikawa, 2004; Crews et al., 2005) of ADG and MWT on DMI. Test group (six levels) was included as an independent variable in the calculation of RFI. In each case, individual RFI was computed as actual DMI minus the expected DMI predicted from the appropriate phenotypic or genetic regression model. Ultrasound backfat thickness (**UBF**), LM area (**ULMA**), and marbling score (**UMAR**) were predicted from linear regression against time of measurements obtained every 28 days with an Aloka 500V real-time ultrasound with a 17 cm, 3.5-MHz linear array transducer (Overseas Monitor Corporation Ltd., Richmond, BC).

The feedlot behaviour traits studied were daily feeding duration (**FD**), daily feeding "head down" time (**HD**), daily feeding frequency (**FF**) and mean flight speed (**FS**; as a measure of temperament). Procedures for determining feeding behaviour from the GrowSafe System have previously been described (Basarab et al., 2003). Daily feeding duration was computed as the sum of the difference between feeding event end-times and start-times per day for each animal. It was equal to the total number of minutes each day spent in feeding-related activities (prehension, chewing, backing away from the bunk and chewing, socializing, scratching, or licking) at the feedbunk. Head down time refers to the sum of the number of times the animal's electronic identification (transponder) was detected by the GrowSafe System during a feeding event multiplied by the system's scanning time, where scanning time is system-dependent and ranges from 1.0 to 6.3 s. attendance. Daily feeding frequency in this study was defined as the number of independent feeding events for a particular animal in a day. A feeding event starts when an animal's transponder is first detected and ends when the time between the last two transponder readings was greater than 300 seconds, the transponder is detected at another bunk, or when a different transponder number is encountered. Flight speed was calculated from the time in seconds taken to traverse a fixed distance of 2.44 m after exiting a squeeze chute. Infrared sensors were used to trigger the start and stop of the timing system (Burrow et al., 1988).

Carcass traits were evaluated according to the Canadian beef carcass grading system (Agriculture Canada, 1992). Carcass weight (**CWT**) of each animal was determined as the weight of the left and right halves of the carcass. Carcass grade fat (**CGF**) was measured at the 12/13th rib of each carcass. Carcass marbling score (**CMAR**) is a measure of intramuscular fat and can be classified as 1 to < 2 units = trace marbling (Canada A quality grade, **QG**); 2 to < 3 units = slight marbling (Canada AA quality grade); 3 to < 4 units =

small to moderate marbling (Canada AAA **QG**) and ≥ 4 units = slightly abundant or more marbling (Canada Prime). Lean meat yield (**LMY**) is an estimate of saleable meat and was calculated according to the equation, lean % = 63.65 + 1.05 (muscle score) - 0.76 (grade fat). Yield grade (**YG**) is the proportion of lean meat and is classified as Y1 = >59 %, Y2 = 54 to 58%, and Y3 < 54%.

5.2.3 Statistical Analyses

There were 464 animals with performance, feed efficiency, feeding behaviour, and ultrasound records, 381 of the 464 animals with carcass merit records, and 302 of the 464 animals with temperament records (Table 5.1). The total number of animals including those without records but contributing pedigree information was 813. The animals in the study were primarily paternal half-sibs, but some dams were used in multiple years on the same sires, making some of the animals full siblings. The steers were classified into high, medium and low RFIp and RFIg groups based on ± 0.5 standard deviations from the mean. This was done in order to determine the differences in behaviour among animals belonging to different classes of feed efficiency.

Differences in feeding behaviour and temperament among steers in different classes of RFIp and RFIg were determined using a SAS mixed model (SAS Institute, Inc., Cary, NC, version 9.1.3), which included fixed effects of RFIp group (high, medium, and low), breed of sire (Angus, Charolais, or Hybrid), test year (three levels), test group nested within year (two groups per year), and random effects of a classification variable of sire and dam of steer. Age on test was included in the model as a linear covariate. Mean separation among RFI groups for different test traits was carried out by least-squares using the PDIFF option in SAS. The PROC CORR of SAS was used to obtain Pearson partial phenotypic correlations adjusted for the linear effects of age and the fixed effect of year.

Genetic (co)variances were obtained with the statistical software ASREML (Gilmour et al. 2000). A preliminary univariate analysis for each trait was carried out to obtain starting (co)variance parameters that were then fitted in subsequent restricted maximum likelihood (REML) bivariate analyses. Pairwise bivariate analyses were performed for each behavioural trait and against the other test traits. The two-trait individual animal model used to estimate (co)variance components included fixed contemporary group effect, random additive genetic and residual effects, and linear covariate for age. Genetic variances and heritability estimates for any particular trait were calculated as the average value of the estimates from all pairwise bivariate analyses performed against all traits, while their standard errors were the medians of the SE estimates.

5.3 Results

The considerable variation in the various growth, feed intake, feed efficiency, ultrasound, and carcass merit traits among the animals (Table 3.2) made it appropriate for determining their relationships with the different measures of behaviour (Table 5.1) in the study. Estimates of heritabilities, genetic and phenotypic relationships among measures of feeding behaviour and temperament are shown in Table 5.2. Heritability estimates for feeding duration, feeding head down time, feeding frequency, and flight speed were 0.28, 0.33, 0.38, and 0.49, respectively. Daily feeding duration was positively correlated phenotypically with head down time ($P < 0.001$) and feeding frequency ($P < 0.05$) but was unrelated phenotypically with flight speed. Head down time had a moderate positive phenotypic correlation with feeding frequency ($P < 0.001$) but tended to have a weak negative correlation with flight speed ($P < 0.10$). Feeding frequency was phenotypically unrelated to flight speed.

Daily feeding duration had a low positive genetic correlation with head down time and a moderate positive genetic correlation with flight speed. Head down time had a positive genetic correlation with feeding frequency and a high negative genetic correlation with flight speed. The genetic correlation of feeding frequency with flight speed was not different from zero. Differences among animals with high, medium or low RFIp and RFIg in feeding behaviour and flight speed are shown in Tables 5.3 and 5.4, respectively, whilst correlations of the different measures of feeding behaviour and temperament with feed intake and different measures of feed efficiency are presented in Table 5.5. Daily feeding duration and head down time had positive phenotypic correlations with DMI, RFIp and RFIg and a negative correlation with PEG, respectively ($P < 0.001$), but were unrelated phenotypically with FCR. Indeed, animals with high genetic and phenotypic RFI consistently had higher feeding durations and head down times, respectively, compared to animals with low RFI (*P* < 0.001). Feeding frequency had a low negative phenotypic correlation with DMI and FCR, but its phenotypic relationships with RFIp, RFIg, and PEG were positive (*P* < 0.05).

The genetic correlations of both feeding duration and head down time with DMI, RFIp, RFIg, and PEG were moderate to high and were in the same direction as the phenotypic relationships. Feeding frequency had a moderate to high negative genetic correlation with DMI, FCR, RFIp and RFIg, but a strong positive relationship with PEG. Flight speed was negatively correlated phenotypically with DMI (*P* < 0.001) and PEG (P < 0.05), but was unrelated phenotypically with FCR, RFIp or RFIg (*P* > 0.10). In fact, flight speed only tended to differ among animals with low or high genetic or phenotypic RFI (*P* = 0.10). On the other hand, flight speed had a weak negative genetic correlation with DMI, a moderate correlation with FCR, and a moderate to strong negative genetic correlation with the other measures of feed efficiency.

The relationships of the different measures of behaviour with growth, BW and ultrasound traits are shown in Table 5.6. Feeding duration had a low positive phenotypic correlation with ADG, BW, ultrasound backfat, LM area, and marbling score (*P* < 0.01). Similarly, feeding head down time was positively phenotypically correlated with ADG and ultrasound backfat (*P* < 0.05), but was unrelated to BW, ultrasound LM area, or marbling score. Feeding frequency had a negative phenotypic correlation with BW and a positive correlation with ultrasound LM area (*P* < 0.05), but was unrelated to ADG, UBF, or UMAR (*P* > 0.10). Flight speed was phenotypically negatively related to ADG and positively related to ultrasound LM area (*P* < 0.01) but was unrelated to BW, ultrasound backfat, or marbling score (*P* > 0.10). Daily feeding duration and head down time had positive genetic correlations with ADG, BW, ultrasound backfat and marbling score, but a negative genetic correlation with ultrasound LM area. The genetic correlations of feeding frequency with ADG, final BW, ultrasound backfat, LM area and marbling score were negative. Flight speed had weak negative genetic correlations with ADG and ultrasound marbling and a strong negative genetic correlation with final BW. In addition, flight speed showed a moderate positive genetic correlation with ultrasound backfat and a strong correlation with ultrasound LM area.

The relationships of the different measures of behaviour with measure of carcass merit are shown in Table 5.7. Daily feeding duration was positively correlated phenotypically with carcass weight, grade fat, LM area, yield grade and marbling score , but was negatively correlated with lean meat yield ($P < 0.01$). Feeding duration was however, unrelated to carcass quality grade ($P > 0.10$). On the other hand feeding head down time was phenotypically unrelated to carcass traits ($P > 0.10$) except a tendency towards a negative correlation with carcass LM area ($P < 0.10$) and a weak positive correlation with carcass quality grade. Feeding frequency was negatively phenotypically correlated with carcass weight, grade fat, marbling score and yield grade and positively phenotypically correlated with lean meat yield and quality grade ($P < 0.05$); feeding frequency was unrelated to carcass LM area. Flight speed was negatively correlated phenotypically with carcass weight, grade fat, marbling score, yield grade, and quality grade, but positively correlated with carcass LM area and lean yield ($P < 0.05$).

The genetic correlations of feeding duration with carcass traits ranged from moderate to high and were positive for carcass weight, grade fat, marbling score, yield grade and lean meat yield; genetic correlations of feeding duration with carcass LM area and lean yield were negative. The genetic correlations of head down time with carcass traits were similar in magnitude and sign to that of feeding duration with these traits, except that head down time was unrelated to marbling score and had a weak correlation with grade fat. With the exception of lean meat yield and quality grade, which had no genetic correlations and a positive genetic correlation with feeding frequency, respectively, the genetic relationships of feeding frequency with the remaining carcass traits were negative and moderate. Flight speed had moderate to high negative genetic correlations with carcass weight, carcass yield grade, and quality grade, and positive genetic correlations with carcass LM area, lean meat yield and marbling score.

5.4 Discussion

Characterization of groups of individual animals into different feeding behaviour and temperament groups would provide useful insights into sources of variation in animal performance, feed efficiency and carcass merit. This information is not only useful for developing appropriate feedbunk management practices, but may also be employed in selection programs to address any potentially adverse correlated responses in behaviour following selection for certain ERT. The mean values for feeding duration and feeding frequency reported in the present study are lower compared to data reported using similar facilities by Basarab et al. (2003) and Schwartzkopf-Genswein et al. (2003). However, the feeding frequencies reported in the present study are similar to those reported by Schwartzkopf-Genswein et al. (2002) (31.0 ± 6.2 vs. 29.6 ± 0.5 events/d). The feeding head down times reported in the present study are comparable to values reported by Basarab et al. (2002) using similar test facilities. Possible reasons for the disparities in magnitude of feeding behaviour traits from the different studies may include differences in the numbers of animals per test (stocking density) as well as possible interactions between feed type and factors such as meal size and bite size. For instance, Basarab et al. (2000) indicated that switching from a growing diet (higher forage content) to a finishing diet (higher concentrate content) in cattle may result in as high as 30% reduction in feeding frequency but with a corresponding increase in feeding duration. Factors such as ambient temperature, relative humidity, barometric pressure and wind speed have also been identified as significantly influencing feeding patterns of cattle tested using the GrowSafe System (Schwartzkopf-Genswein et al., 2003).

Linear regression analysis indicated that approximately 55% of the daily feeding duration may actually be related to feed consumption (feeding head down time), whilst the remainder may be spent in other feeding related activities at the feedbunk. The direct heritability estimates for feeding duration and feeding frequency reported in the present study were comparable to values reported for ram lambs (0.35 – 0.36, Cammack et al., 2005). In addition, the estimates obtained in the present study are somewhat lower than those reported by Robinson and Oddy (2004) for daily feeding duration (0.36) and daily feeding sessions (0.44) for tropically adapted or temperate heifers and steers. The phenotypic correlation between feeding duration and feeding frequency reported in this study tended to be lower than that reported in ram lambs (0.25, Cammack et al., 2005). The negative genetic correlation between feeding duration and feeding frequency reported in the present study are in contrast to the strong positive relationship reported by Cammack et al. (2005).

The flight speed values reported in the present study are consistent with values reported by Burrow and Corbet (2000) for calves born to a range of *Bos taurus and Bos indicus* sires as well as values reported by Curley et al. (2004) for Brahman heifers. The direct heritability estimate for flight speed reported in the present study tended to be higher than that reported (0.35) by Burrow and Corbet (2000), but was more like that reported (0.40 - 0.44) by Burrow (2001). No report was located in the literature comparing a measure of temperament with feeding behaviour. The results obtained in this study indicate however, that even though feeding behaviour may be phenotypically independent of temperament, the two classes of behaviour traits may not be necessarily genetically independent (considering only the point estimates of genetic correlation). The positive genetic correlation between feeding duration and temperament may indicate a commonality in the genetics of the two traits, whereas there may be an inverse relationship between the genetic factors that affect temperament and those directly related to feed consumption. This is not only evident from the negative genetic correlation between flight speed and head down time, but also from the phenotypic and genetic correlations between flight speed and DMI.

The phenotypic relationship of feeding duration with feed intake in this study were similar to that found by Schwartzkopf-Genswein et al. (2002) and Robinson and Oddy (2004), who observed a moderate positive relationship (r = 0.30) between feed intake and feeding duration. The results suggest that the longer animals spend at the bunk, the more feed they consumed. Basarab et al. (2002) looked at the relationship between feed intake and feeding behaviour traits in feedlot cattle. Their results indicated that mean correlation coefficients were 0.69, 0.59 and 0.31 for the relationship between daily feed intake and daily feeding duration, head down time and frequency, respectively. These results are very similar to those of the present study and suggest that daily feeding duration had the largest, positive relationship to daily feed intake, while feeding frequency was only moderately related to feed intake. Cammack et al. (2005) reported lower genetic and phenotypic correlations between feed intake and feeding duration. The negative phenotypic and genetic correlations between DMI and feeding frequency reported in this study were higher and in contrast to the correlations reported by Cammack et al. (2005). Additionally, these results are contrasting to the findings of Robinson and Oddy (2004) who observed a weak positive phenotypic correlation (r = 0.18) but no genetic correlation between feed intake and daily feeding duration.

Generally, the relationships between the measures of feeding behaviour with RFI and PEG were higher than those with FCR and DMI. Schwartzkopf-Genswein et al. (2002) observed a negative relationship between feeding duration and FCR (r = -0.17). In the present study, animals with low phenotypic RFI had 24% and 14% lower feeding duration, 29% and 18% lower head down time as well as 14% and 10% lower feeding frequency compared to animals with high or medium phenotypic RFI, respectively. Very similar relationships were observed between genetic RFI and the feeding behaviour traits. The relationships of RFI with feeding duration and feeding frequency were also observed by Basarab et al. (2003), though in the study by these authors, the differences observed between different RFI classes and both feeding behaviour traits were not significant. Cammack et al. (2005) reported positive genetic and phenotypic correlations between RFI and feeding duration and feeding frequency, though their correlation coefficients were lower than observed in this study. Robinson and Oddy (2004) reported weak positive phenotypic correlations between RFIp and daily feeding duration and feeding sessions and moderate positive genetic correlations of daily feeding duration and feeding sessions with RFIp. In the same study, phenotypic correlations of feeding behaviour traits with FCR were not different from zero, but the corresponding genetic correlations were high.

The relationship of feeding duration with ADG and final BW observed in the present study contrast with the findings of Streeter et al. (1999) who reported a negative relationship between feeding duration and ADG. The results are however, consistent with the findings of Schwartzkopf-Genswein et al. (2002), who reported a weak but significant positive phenotypic correlation between ADG and feeding duration (r = 0.14). In the same study, Schwartzkopf-Genswein et al. (2002) reported that low gaining Holstein steers had higher feeding durations, though not significantly so; the opposite of this relationship was observed in Charolais steers. In addition, Robinson and Oddy (2004) reported a weak positive but significant phenotypic correlation between feeding duration and ADG, though the corresponding genetic correlation was not different from zero. The present study did not find any phenotypic correlation between ADG and feeding frequency, and the genetic correlation between the two traits was negative. This is in contrast to the findings of Schwartzkopf-Genswein et al. (2003) in cattle and that of Cammack et al. (2005) who reported positive correlations between ADG and feeding frequency. Similarly, Robinson and Oddy (2004) reported weak but significant positive phenotypic correlation but no genetic correlation between ADG and daily feeding sessions.

It must be noted that the number of animals per test and the magnitude of the feeding behaviour traits reported in the present study are considerably different from the studies reported by Schwartzkopf-Genswein et al. (2003) and Cammack et al. (2005). In the present study, there were approximately 80 animals feeding from ten feedbunks. The observed relationships of ADG and BW with the feeding behaviour traits indicate that bigger animals spent more time at the feed bunk. It is possible that a large proportion of this time may not be associated with actual feed consumption but was a possible means to maintain their social dominance at the bunk. As such, these bigger animals may visit the feed bunk less frequently and stay longer on each visit, possibly to maintain or take advantage of their social position.

The relationship of flight speed with DMI and ADG in the present study are consistent with the findings of Fox et al. (2004) who observed negative phenotypic correlations between flight speed and DMI (r = -0.34, -0.17) and ADG (r = -0.25, -0.25) for Bonsmara bulls and Santa Gertrudis steers, respectively. In addition, Burrow and Dillon (1997) reported that animals with slow flight speeds gained weight more rapidly and had heavier slaughter weights than animals with fast flight speeds in *Bos indicus* crossbred cattle. Also, Voisinet et al. (1997) observed that cattle that became agitated during handling had 14% lower weight gains compared to calmer animals. On the contrary, Burrow (2001) did not detect any phenotypic or genetic relationships between flight speed and birth weight, weaning weight, yearling weight, final BW, or ADG in a tropically adapted composite breed of cattle grazed at pasture in the tropics. The lack of phenotypic correlation of flight speed with FCR and RFI were consistent with the findings of Fox et al. (2004) who did not observe any correlations between flight speed and net feed efficiency. However, the present study detected moderate to strong genetic correlations between measures of feed efficiency and flight speed, indicating that there may be a need to include temperament in any genetic selection programs involving feed efficiency in order to overcome potential problems of poor temperament in efficient animals.

The phenotypic correlations of measures of feeding behaviour with ultrasound and carcass traits were mostly low but generally different from zero ($P < 0.05$). The corresponding genetic correlations were higher than the phenotypic correlations. There are very few reports in the literature relating measures of feeding behaviour with ultrasound and carcass traits, and the relevance of the relationships obtained in the present study are yet to be determined. Robinson and Oddy (2004) observed that the phenotypic correlations of feeding behaviour traits with ultrasound intramuscular fat, rump fat, rib fat, and LM area were not different from zero. The same authors showed that daily feeding duration had a low negative genetic correlation with ultrasound intramuscular fat and a moderate positive genetic correlation with LM area. Robinson and Oddy (2004) also reported that daily feeding sessions had a weak positive genetic correlation with intramuscular fat, weak negative genetic correlation with rib fat, and a moderate negative genetic correlation with LM area. Thus the relationships between daily feeding frequency and duration with ultrasound or carcass marbling score and LM area are consistent with the results of the present study, albeit some differences in the strength of the correlation coefficients.

The relationships between flight speed and carcass weight in the present study are consistent with that of Voisinet et al. (1997) who observed that cattle that became agitated during handling had 10% lower carcass weights compared to calmer animals. In addition, Burrow and Dillon (1997) reported that *Bos indicus* crossbred cattle with slow flight speeds had heavier carcass weights than animals with fast flight speeds. The relationships of the behaviour traits with the carcass traits generally reflected the relationships between growth rate, feed intake and body composition. Animals that spend more time eating in a day generally had fatter carcasses, whereas those that spent less time eating had leaner carcasses but lower ADG. Relationship of marbling with feeding behaviour may be due to the correlated indirect relationship with body fatness in general. In conclusion, there is considerable genetic and phenotypic variation in cattle in measures of feeding behaviour and temperament, which are also related to (and may be predictive of) some measures of performance, feed efficiency, and carcass merit. The genetic and phenotypic relationships of these traits need to be given serious consideration in any program to select animals for improved feed efficiency.

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Table 5.1. Number of animals, overall mean, SD, and SE of the behaviour traits considered in the present study

Traits	Animals	Mean	SD	SE
Head down, min/d	464	36.57	11.91	0.55
Feeding frequency, events/d	464	29.62	10.19	0.47
Feeding duration, min/d	464	66.09	18.61	0.86
Flight speed, m/s	302	2.52	0.73	0.04

Table 5.2. Heritabilities (± SE), Genetic (below diagonal ± SE), and phenotypic relationships (above diagonal) of feeding behaviour and temperament in beef cattle.

Traits[a]	FD	HD	FF	FS
FD	**0.28 ± 0.12**	0.64***	0.15*	-0.03
HD	0.25 ± 0.32	**0.33 ± 0.12**	0.45***	-0.11†
FF	-0.40 ± 0.30	0.47 ± 0.24	**0.38 ± 0.13**	0.08
FS	0.42 ± 0.26	-0.56 ± 0.38	-0.11 ± 0.30	**0.49 ± 0.18**

[a] FD = daily feeding duration (min/d); HD = daily feeding head down time (min/d); FF = daily feeding frequency (events/d); FS = flight speed (m/s).

† ($P < 0.10$); * ($P < 0.05$); *** ($P < 0.001$).

Table 5.3. Least squares mean (± SE) for feeding behaviour and temperament in animals differing in phenotypic residual feed intake (RFIp).

Traits	Phenotypic residual feed intake group[a]			P value[b]
	High	Medium	Low	
Number of animals	139	183	142	
Phenotypic RFI, kg DM/d	1.00 ± 0.04[d]	-0.01 ± 0.03[e]	-0.96 ± 0.03[f]	<0.001
Feeding duration, min/d	74.62 ± 1.39[d]	65.64 ± 1.22[f]	56.41 ± 1.35[f]	<0.001
Head down, min/d	42.37 ± 1.18[d]	37.06 ± 1.06[e]	30.28 ± 1.16[f]	<0.001
Feeding frequency, events/d	31.50 ± 0.54[d]	30.36 ± 0.46[e]	27.24 ± 0.51[f]	<0.001
Flight speed[c], m/s	2.43 ± 0.08[e]	2.50 ± 0.07[de]	2.66[d] ± 0.09	0.10

[a] Residual feed intake groups are defined as High, RFI is > 0.5 SD above the mean; Medium, RFI is within ± 0.5 SD of the mean; Low, RFI is < -0.5 SD below the mean.

[b] P values from overall F test.

[c] Animals in high, medium, and low RFI groups were 98, 116 and 88, respectively

[d, e, f] Within a row, means without a common superscript letter differ ($P < 0.05$).

Table 5.4. Least squares mean (± SE) for feeding behaviour and temperament in animals differing in genetic residual feed intake (RFIg).

Traits	Genetic residual feed intake group[a]			P value[b]
	High	Medium	Low	
Number of animals	119	177	168	
Genetic RFI, kg DM/d	1.07 ± 0.05[d]	-0.06 ± 0.04[e]	-1.12 ± 0.04[f]	<0.001
Feeding duration, min/d	74.67 ± 1.57[d]	65.59 ± 1.30[e]	58.50 ± 1.41[f]	<0.001
Head down, min/d	43.14 ± 1.31[d]	36.84 ± 1.12[e]	31.94 ± 1.19[f]	<0.001
Feeding frequency, events/d	31.58 ± 0.59[d]	30.54 ± 0.45[e]	27.57 ± 0.50[f]	<0.001
Flight speed[c], m/s	2.46 ± 0.08	2.49 ± 0.07	2.69 ± 0.10	0.10

[a] Residual feed intake groups are defined as High, RFI is > 0.5 SD above the mean; Medium, RFI is within ± 0.5 SD above and below the mean; Low, RFI is < -0.5 SD below the mean.

[b] P values from overall F test.

[c] Animals in High, Medium, and Low RFI groups were 106, 131 and 65, respectively

[d, e, f] Within a row, means without a common superscript letter differ ($P < 0.05$).

Table 5.5. Genetic (± SE) and phenotypic relationships of feeding behaviour and temperament with feed intake and feed efficiency in beef cattle

Traits[a]	DMI	FCR	RFIp	RFIg	PEG
Phenotypic correlations					
FD	0.27***	-0.06	0.49***	0.41***	-0.44***
HD	0.33***	0.07	0.50***	0.45***	-0.43***
FF	-0.21***	-0.13*	0.18**	0.16**	0.27***
FS	-0.35***	0.03	-0.07	-0.03	-0.13*
Genetic correlations					
FD	0.56 ± 0.20	-0.25 ± 0.29	0.57 ± 0.28	0.43 ± 0.24	-0.61 ± 0.23
HD	0.59 ± 0.21	-0.46 ± 0.32	0.33 ± 0.30	0.42 ± 0.40	-0.45 ± 0.34
FF	-0.74 ± 0.15	-0.52 ± 0.21	-0.34 ± 0.30	-0.77 ± 0.21	0.59 ± 0.23
FS	-0.11 ± 0.26	0.40 ± 0.26	-0.59 ± 0.45	-0.44 ± 0.23	-0.72 ± 0.19

[a] FD = daily feeding duration (min/d); HD = daily feeding head down time (min/d); FF = daily feeding frequency (events/d); FS = flight speed (m/s); FCR = feed conversion ratio (kg DM/kg gain); RFIp = phenotypic residual feed intake (kg DM/d); RFIg = genetic residual feed intake (kg DM/d); PEG = partial efficiency of growth

* ($P < 0.05$); ** ($P < 0.01$); *** ($P < 0.001$).

Table 5.6. Genetic and phenotypic relationships of feeding behaviour and temperament with growth rate, BW and ultrasound measurements in beef cattle

Traits[a]	ADG	FWT	UBF	ULMA	UMAR
Phenotypic correlations					
FD	0.25**	0.32***	0.37***	0.21**	0.25**
HD	0.18**	0.06	0.14*	0.08	0.04
FF	-0.04	-0.19**	0.05	0.17**	-0.09
FS	-0.26**	-0.08	-0.03	0.22**	-0.08
Genetic correlations					
FD	0.42 ± 0.25	0.46 ± 0.24	0.37 ± 0.25	0.35 ± 0.26	0.59 ± 0.22
HD	0.45 ± 0.26	0.12 ± 0.26	0.16 ± 0.27	-0.62 ± 0.25	0.21 ± 0.23
FF	-0.33 ± 0.23	-0.41 ± 0.22	-0.47 ± 0.22	-0.73 ± 0.18	-0.26 ± 0.21
FS	-0.25 ± 0.25	-0.57 ± 0.29	0.36 ± 0.23	0.81 ± 0.16	-0.13 ± 0.23

[a] FD = daily feeding duration (min/d); HD = daily feeding head down time (min/d); FF = daily feeding frequency (events/d); EV = flight speed (m/s); FWT = final BW (kg); UBF = ultrasound backfat thickness (mm), ULMA = ultrasound LM area (cm^2); UMAR = ultrasound marbling score.

* ($P < 0.05$); ** ($P < 0.01$); *** ($P < 0.001$).

Table 5.7. Genetic and phenotypic relationships of feeding behaviour and temperament with measures of carcass merit in beef cattle

Traits[a]	CWT	CGF	CLMA	LMY	CMAR	YG
			Phenotypic correlations			
FD	0.27***	0.23**	0.15*	-0.18**	0.15*	0.23**
HD	0.09	-0.04	-0.10†	-0.06	-0.05	0.03
FF	-0.24**	-0.26***	0.08	0.17**	-0.22**	-0.24**
FS	-0.25**	-0.25**	0.14*	0.30***	-0.22**	-0.25**
			Genetic correlations			
FD	0.31 ± 0.29	0.38 ± 0.26	0.62 ± 0.24	0.62 ± 0.21	0.56 ± 0.27	0.67 ± 0.21
HD	0.29 ± 0.28	0.13 ± 0.28	-0.41 ± 0.27	-0.30 ± 0.24	0.04 ± 0.28	0.41 ± 0.24
FF	-0.33 ± 0.27	-0.47 ± 0.23	-0.76 ± 0.19	0.04 ± 0.22	-0.42 ± 0.23	-0.34 ± 0.25
FS	-0.54 ± 0.32	-0.11 ± 0.27	0.32 ± 0.25	0.33 ± 0.23	0.10 ± 0.28	-0.22 ± 0.27

[a] FD = daily feeding duration (min/d); HD = daily feeding head down time (min/d); FF = daily feeding frequency (events/d); EV = flight speed (m/s); CWT = carcass weight (kg); CGF = carcass grade fat (mm), CLMA = carcass LM area (cm^2); LMY = carcass lean meat yield (%); CMAR = carcass marbling score; YG = carcass yield grade; and QG = carcass quality grade.

† ($P < 0.10$); * ($P < 0.05$); ** ($P < 0.01$); *** ($P < 0.001$).

CHAPTER 6

Genetic and Phenotypic Relationships of Serum Leptin Concentration with Performance, Feed Efficiency, Ultrasound, and Carcass Merit of Composite Beef Cattle.

6.1 Introduction

Leptin is an adipocyte-derived cytokine product of the obese gene that circulates in the serum in the free and bound forms (Zhang et al., 1994). It functions, through interactions with the leptin receptor in the hypothalamus, as a regulator of BW, feed intake, energy expenditure (Houseknecht et al., 1998; Woods et al., 1998), reproduction (Garcia et al., 2002) and immune system functions (Lord et al., 1998). Circulating leptin or tissue mRNA levels are correlated with body weight, food intake, and body fatness in humans or animals (Larsson et al., 1998; Delavaud et al., 2002; Berg et al., 2003). In cattle, circulating leptin levels are correlated with the regional distribution of body fat (Yamada et al., 2003) and carcass merit (Minton et al., 1998; Geary et al., 2003). In addition, mutations in the leptin gene or its promoter have been shown to be associated with serum leptin concentration (Buchanan et al., 2002; Liefers et al., 2003; Nkrumah et al., 2005; Schenkel et al., 2005) and certain economically relevant traits (**ERT**) in beef and dairy cattle.

A number of studies have been carried out to determine the role of circulating leptin on ERT in cattle (Ehrhardt et al., 2000; Delavaud et al., 2002; Garcia et al., 2002). However, little is known about the genetic and phenotypic relationships of endocrine leptin with the performance or efficiency traits in farm animals (Berg et al., 2003; Richardson et al., 2004). These and other previous studies mostly involved data on relatively fewer animals, animals under fasting or underfeeding treatments, or following leptin administration. Additionally, there are no genetic parameter estimates for the reported (Minton et al., 1998; Geary et al., 2003) relationships of circulating leptin with the carcass merit of beef cattle. The objective of this study was to determine the genetic and phenotypic relationship of serum leptin concentration with performance, feed efficiency, and measures of ultrasound and carcass merit using data on crossbred composite beef cattle.

6.2 Materials and Methods

6.2.1 Animals and Management

Growth, feed intake, feeding behaviour, temperament, ultrasound and carcass data were collected on 464 composite beef steers sired by Angus, Charolais or University of Alberta Hybrid bulls between 2002 and 2005. The dams used were produced from crosses among three composite cattle lines, namely Beef Synthetic 1 (BS1), Beef Synthetic 2 (BS2) and Dairy x Beef Synthetic (DBS) (Goonewardene et al., 2003). Cows and heifers for the study were bred in multiple sire breeding groups on pasture and the sire of each calf was later determined in a parentage test using a panel of bovine microsatellite markers. The animals were managed and tested under feedlot conditions using the GrowSafe automated feeding system (GrowSafe Systems Ltd., Airdrie, Alberta, Canada) at the University of Alberta's Kinsella Research Station. Details of the procedures for the feedlot tests were given by Nkrumah et al. (2004) and are as indicated in Chapter 3. The animals weighed 353.0 (SD = 61.3) kg and were 252 (SD = 42) days of age at the beginning of tests. Two tests made up of approximately 80 animals per test were conducted each year. The animals used in the study were cared for according to the guidelines of the Canadian Council on Animal Care (CCAC, 1993).

In yr one, steers were fed free choice a backgrounding diet of mainly alfalfa-brome hay with oats supplemented with corn grain and feedlot mineral supplement to promote a growth rate of just under 1.0 kg/d for approximately 30 d. This period was followed by a 30-d pre-test adjustment period in which the amount of corn in the backgrounding diet was adjusted up gradually to introduce the animals to the test diet and the feeding system. This was done to allow them to adapt to the diet and learn to feed from the test facility. The test diet in yr one was composed of 80.0% dry-rolled corn, 13.5% alfalfa hay pellet, 5% feedlot supplement (32% CP beef supplement) and 1.5% canola oil, supplying approximately 2.90 Mcal/kg of ME and 12.5% CP. In yr two and three, the same test procedures were used, but the test diet contained 64.5% barley grain, 20% oat grain, 9.0% alfalfa hay pellet, 5.0% beef feedlot supplement and 1.5% canola oil, supplying 14.0% CP and 2.91 Mcal/kg of ME. Corn was used in yr 1, instead of barley and oats, because of a feed barley shortage that particular year.

6.2.2 *Blood collection and leptin assay*

At the end of the feed efficiency tests (one week prior to slaughter), blood samples were collected from each animal by jugular venipuncture into Vacutainer tubes. Animals were bled in the morning and all animals were allowed unrestricted access to feed and water prior to bleeding. Blood samples were allowed to clot for approximately 18 h at 4°C. Samples were centrifuged at 2,500g for 30 minutes, and serum collected and stored at -20°C until assayed for leptin using the leptin radio-immuno assay (RIA) kit described by Delavaud et al. (2000). Intra- and interassay coefficients of variation for the leptin assays were less than 5%. Standard deviations above and below the mean leptin concentration were used to group animals into high serum leptin (> 0.5 SD above the mean), medium serum leptin (within ± 0.5 SD of the mean) and low serum leptin (< 0.5 SD below the mean). At the end of the feed efficiency tests animals were weighed and shipped to a commercial packing plant where they were harvested the following day and standard industry carcass data collected after a 24-h chill at -4°C.

6.2.3 *Traits analyzed and their derivations*

Procedures for obtaining the measures of feedlot performance and feed efficiency have been described previously (Nkrumah et al., 2004). Linear regression in SAS (SAS Institute, Inc., Cary, NC, version 9.1.3) of weekly or fortnightly BW against time (d) was used to derive ADG, final BW and mid-test metabolic size (**MWT**, $BW^{0.75}$) for each steer. The total feed intake of each animal over a 70-d test period was used to compute the daily DMI. Feed conversion ratio (**FCR**) was computed as the ratio of DMI to ADG on test. The partial efficiency of growth (PEG; i.e., energetic efficiency for ADG above maintenance) of each animal was computed as the ratio of ADG to the difference between average daily DMI and expected DMI for maintenance (**DMIm**) (Arthur et al., 2001), where DMIm was computed according to NRC (1996).

Residual feed intake (**RFI**, an alternative measure of feed efficiency) was calculated both from phenotypic regression (**RFIp,** Arthur et al., 2001) or genetic regression (**RFIg,** Crews et al., 2005; Hoque and Oikawa, 2004) of ADG and MWT on DMI. Test group (six levels) was included as an independent variable in the calculation of RFI. In each case, individual RFI was computed as actual DMI minus the expected DMI predicted from the appropriate phenotypic or genetic regression model. Ultrasound backfat thickness (**UBF**), LM area (**ULMA**), and marbling score (**UMAR**) were predicted from linear regressions against time of measurements obtained every 28 days with an Aloka 500V real-time ultrasound with a 17 cm, 3.5-MHz linear array transducer (Overseas Monitor Corporation Ltd., Richmond, BC).

Carcass traits were evaluated according to the Canadian beef carcass grading system (Agriculture Canada, 1992). Carcass weight (**CWT**) of each animal was determined as the weight of the left and right halves of the carcass. Carcass grade fat (**CGF**) was measured at the 12/13th rib of each carcass. Carcass marbling score (**CMAR**) is a measure of intramuscular fat and can be classified as 1 to < 2 units = trace marbling (Canada A quality grade, **QG**); 2 to < 3 units = slight marbling (Canada AA quality grade); 3 to < 4 units = small to moderate marbling (Canada AAA **QG**) and ≥ 4 units = slightly abundant or more marbling (Canada Prime). Lean meat yield (**LMY**) is an estimate of saleable meat and was calculated according to the equation, lean % = 63.65 + 1.05 (muscle score) - 0.76 (grade fat). Yield grade (**YG**) is the proportion of lean meat and is classified as Y1 = >59 %, Y2 = 54 to 58%, and Y3 < 54%.

6.2.4 Statistical Analyses

Performance, feed efficiency and ultrasound data from 464 animals and carcass data from 381 animals were analyzed. Effects of breed of sire or serum leptin group on leptin concentration, growth, feed intake, feed efficiency, ultrasound traits and carcass merit were analyzed by least squares procedures using PROC MIXED of SAS (SAS Institute, Inc., Cary, NC, version 9.1.3). The statistical model used included fixed effects due to leptin group (high, medium and low), breed of sire (Angus, Charolais, and Hybrid), year of test (three levels), test group nested within year (two levels per year), all possible interactions, and linear and quadratic effects of age when $P < 0.05$. All interaction terms that did not account for a significant portion of the observed variance ($P > 0.10$) were subsequently excluded from the final model. The random effects of the models were a class variable of sire and dam and residual effects of animal. The error term for breed of sire was animal within sire. The PROC CORR of SAS was used to obtain Pearson partial phenotypic correlations adjusted for the linear (and sometimes quadratic) effects of age and the fixed effect of year.

Genetic (co)variances were obtained with the statistical software ASREML (Gilmour et al. 2000). A preliminary univariate analysis for each trait was carried out to obtain starting co(variance) parameters that were then fitted in subsequent restricted maximum likelihood (REML) bivariate analyses. Pairwise bivariate analyses were performed between serum leptin concentration and each test trait. The two-trait individual animal model used to estimate (co)variance components included fixed contemporary group effects, random additive genetic and residual effects, and linear or quadratic covariate for age. Similar bivariate analyses were carried out for the relationships among growth, BW and carcass merit traits. Genetic variances and heritability estimates for any particular trait were calculated as the average value of the estimates from all pairwise bivariate analyses performed against all traits and their SE were the medians of the SE estimates.

6.3 Results

The heritability estimate (± SE) for serum leptin concentration in the study was moderate (0.34 ± 0.13). Serum leptin concentration averaged 13.91 (SD = 5.74) ng/mL and ranged from 2.19 to 39.70 ng/mL. This gave a desirable range for comparing serum leptin concentration to the different performance, feed efficiency and carcass traits studied. Table 6.1 shows the differences in serum leptin concentration and measures of growth, BW, ultrasound and carcass traits for animals of different sire breeds. Serum leptin concentration was 20% and 19% lower ($P < 0.001$) for animals sired by Charolais- compared to Angus- and Hybrid-sired animals, respectively. Phenotypic RFI was 0.31 kg and 0.34 kg lower ($P = 0.03$) for Charolais- compared to Angus- and Hybrid-sired cattle ($P = 0.03$).

On the other hand, genetic RFI was 0.23 kg and 0.29 kg lower for Charolais- compared to Angus- and Hybrid-sired cattle. Ultrasound backfat thickness was 27% and 22% lower ($P < 0.001$) for Charolais compared to Angus and Hybrid cattle. Similarly, carcass grade fat was 31 % and 26% lower ($P < 0.001$) for Charolais-sired animals compared to Angus- or Hybrid-sired animals, respectively. Ultrasound marbling score was

14% and 13% lower ($P < 0.01$) in Charolais- than in Angus- or Hybrid-sired cattle, respectively. Carcass marbling score was 15% and 14% lower ($P < 0.001$) in Charolais- than in Angus- or Hybrid-sired cattle, respectively.

Ultrasound LM area tended to be lower ($P = 0.09$) and carcass LM area was lower ($P = 0.03$) in Angus- and Hybrid- compared to Charolais-sired animals, respectively. Carcass lean meat yield was lower ($P < 0.001$) in Angus- and Hybrid-sired animals compared to Charolais-sired animals. Consistently, carcass yield grade was lower (preferred) ($P < 0.01$) in Charolais- compared to Angus- or Hybrid-sired animals. Average daily gain, metabolic BW, final BW, daily DMI, and feed conversion ratio did not differ ($P > 0.10$) among animals from different sire breeds. The genetic and phenotypic relationships of serum leptin concentration and differences among animals with low, medium and high serum leptin concentration in feed intake and measures of feed efficiency are presented in Table 6.2. Daily DMI ($P < 0.01$), RFIp and RFIg ($P < 0.05$) were higher for animals with high serum leptin concentration than for those with low serum leptin concentration.

Consistently, PEG was lower in high serum leptin than in low serum leptin animals. No differences in FCR were observed among the animals differing in serum leptin concentration. The genetic correlations observed for these traits were generally higher than the corresponding phenotypic correlations. Daily DMI had a positive phenotypic correlation ($P < 0.01$) with serum leptin concentration, but the corresponding genetic correlation was negative. The phenotypic correlation of serum leptin concentration with FCR, phenotypic RFI, genetic RFI, and PEG were not different from zero ($P > 0.10$). Genetically, serum leptin concentration was negatively correlated with FCR, RFIp, and RFIg but positively correlated with PEG. The relationships of serum leptin concentration with growth rate, BW, and ultrasound measurements are presented in Tables 6.3 and 6.4.

The phenotypic and genetic correlations among the growth, BW and ultrasound traits observed in the study were significantly different from zero. Steers with high serum leptin concentration had higher metabolic BW, final BW, and slaughter weight compared to those with low leptin concentrations ($P < 0.05$). Metabolic BW ($P < 0.05$) and slaughter weight ($P < 0.10$) had positive phenotypic correlations but negative genetic correlations with serum leptin concentration. Serum leptin concentration was not correlated both genetically and phenotypically with ADG, and there were no differences ($P > 0.10$) in ADG among animals with different leptin concentrations. Ultrasound backfat had a moderate positive ($P < 0.001$) phenotypic correlation and strong positive genetic correlation with serum leptin concentration. Similarly, ultrasound marbling score had positive phenotypic ($P < 0.01$) as well as genetic correlations with serum leptin concentration.

Consistently, animals with high serum leptin had higher ultrasound backfat ($P < 0.001$) and marbling score ($P < 0.01$) compared to animals with low serum leptin concentration; ultrasound backfat had a moderate phenotypic correlation and a strong genetic correlation with ultrasound marbling score in the study. Ultrasound LM area showed a weak negative, but significant ($P < 0.05$) phenotypic correlation with serum leptin concentration, though differences in LM area among animals with different serum leptin was not significant ($P > 0.10$); serum leptin however showed a very strong negative genetic correlation ultrasound LM area.

The relationships of serum leptin concentration with carcass traits are presented in Tables 6.5 and 6.6. Animals with high serum leptin concentration had significantly higher grade fat ($P < 0.001$), marbling ($P < 0.01$), yield grade ($P < 0.001$), and quality grade ($P < 0.001$), compared to animals with low serum leptin concentration. Carcass grade fat, marbling score and yield grade had moderate phenotypic correlations but moderate to strong genetic correlations with serum leptin concentration. Serum leptin concentration was however negatively correlated phenotypically ($P < 0.001$) and genetically with carcass lean meat yield. Animals with high and medium serum leptin concentrations had lower lean meat yield compared to those with low serum leptin concentration. In addition, Carcass LM area had a weak but significant ($P < 0.05$) negative phenotypic correlation with serum leptin concentration; the corresponding genetic correlation was very strong and negative as well.

Carcass weight was not correlated to serum leptin concentration ($P > 0.10$) and did not differ among the different classes of leptin. The heritability estimates for the growth, ultrasound and carcass traits presented in the study were moderate to high. Phenotypically, ultrasound backfat thickness and marbling score were correlated with each other ($P < 0.001$) but were not correlated with LM area ($P > 0.10$). In addition, carcass grade fat and carcass marbling score were phenotypically correlated with each other ($P < 0.001$) and were respectively negatively correlated with lean meat yield ($P < 0.001$) and carcass LM area ($P < 0.01$). The genetic correlation of ultrasound backfat with ultrasound marbling score in the study was high. Ultrasound backfat was not correlated genetically with ultrasound LM area. However, ultrasound marbling had a low negative genetic correlation with ultrasound LM area. The genetic correlations among the various carcass traits in the study were generally moderate to strong, with the exception of the genetic correlations of carcass LM area and lean meat yield with carcass weight which were weak (LM area) or not different from zero (lean meat yield).

6.4 Discussion

The serum leptin concentrations reported in the present study are within the range of values reported by Geary et al. (2003) for composite steers and heifers and Richardson et al. (2004) for Angus steers using the same RIA kit. The present study demonstrated considerable breed differences in serum leptin concentration associated with differences in ultrasound and carcass grade fat, marbling score, yield grade and lean meat yield. Berg et al. (2003) observed that breed differences in serum leptin concentration in pigs were related to growth and carcass characteristics. Ren et al. (2002) reported that plasma leptin tended to be higher and body fat content, omental and perirenal fat mass, as well as leptin mRNA expression in subcutaneous and perirenal fat depots were significantly higher in German Holsteins compared to Charolais. Their results also showed that body weight and LM area were significantly higher in Charolais compared to Holsteins. Thomas et al. (2002) showed that serum leptin levels were higher in Angus bulls compared to their Brahman counterparts of similar ages. Similarly, Yonekura et al. (2002) showed that mean serum leptin concentration was higher in Japanese Black cattle compared to their Holstein counterparts.

Breed differences in circulating leptin levels seem to correlate well with the differential abilities of the breeds to accrete body fat or lean. Wegner et al. (2001) indicated differences in plasma leptin concentration among crossbred cattle varying in percentage Wagyu breed composition. A recent study by Geary et al. (2003) however showed no significant differences in serum leptin concentration among different composite breeds of cattle, despite considerable differences observed among the breeds in measures of body fatness. The breed differences in serum leptin concentration observed in the present study, as well as in most of the studies cited above were considerably lower in magnitude when compared with the differences in ultrasound and carcass merits observed between the same animals. It appears that only part of the breed differences in body composition are related to serum leptin concentration, and other factors may be involved as well. It has been suggested that genetically leaner cattle may be expressing leptin from sites other than adipocytes or their level of expression per gram of adipose tissue may be higher (Geary et al., 2003).

The present study is the first of its kind reporting genetic parameter estimates on the relationships of serum leptin with performance, feed efficiency and carcass quality. The phenotypic relationships between serum leptin concentration and ultrasound and carcass measures of body fatness obtained in the present study were generally in agreement with data published by others (Minton et al., 1998, Geary et al., 2003). Serum leptin concentration also shows significant positive correlations with measures of body fatness in humans (Larsson et al., 1998) with percent body fat content of 35.6 ± 3.6 vs. 27.4 ± 2.9 in humans with high vs. low serum leptin, respectively. It is therefore surprising that a number of studies (Kawakita et al., 2001, Yonekura et al., 2002) involving Japanese Black cattle failed to demonstrate a relationship between body fatness and plasma leptin concentration.

Kawakita et al. (2001) attributed the differences in findings using other cattle compared to Japanese Black cattle to the greater ability of the latter breed to deposit intramuscular fat (Zembayashi, 1994; Zembayashi, 1995). This is however in contrast to the findings of Wegner et al. (2001) who observed significant correlations between plasma leptin concentration and muscle lipid content in crossbred Wagyu cattle. The study by Wegner et al. (2001) indicated that correlations of plasma leptin concentration with LM lipid content for 0%, 50% and 75% Wagyu cattle were r = 0.62, r = 0.11 and r = -0.60, respectively. It appears that the superior ability of Japanese Black cattle to deposit intramuscular fat somehow alters the relationship of circulating leptin with body fatness in that the relationship of circulating leptin is stronger with subcutaneous fat level than with intramuscular fat level.

The genetic parameter estimates as well as the relationships among the measures of growth, BW and different measures of ultrasound and carcass quality observed in the present study are generally consistent with those reported in previous studies (Bertrand et al., 2001; Crews et al., 2003; Devitt and Wilton, 2001). The relationship of serum leptin concentration with ultrasound and carcass LM area observed in the present study are conflicting with the results of Wegner et al. (2001) who observed that plasma leptin concentration was unrelated to LM area in crossbred Wagyu cattle. Berg et al. (2003) also observed a negative correlation (r = -0.33) between LM area and serum leptin in pigs. Geary et al. (2003) showed no significant phenotypic relationships of LM area with serum leptin concentration in one study consisting of a group of crossbred composite steers composed predominantly of Angus; but showed a negative relationship (r = -0.45) between LM area and serum leptin in a separate group consisting of steers and heifer progeny of the composite breed above. Additionally, Minton et al. (1998) showed a positive relationship (r = 0.32) between LM area and serum leptin concentration. These contrasting correlations between LM area and serum leptin seem to be highly reflective of the published correlations of LM area and marbling score or subcutaneous fat thickness in cattle (Bertrand et al., 2001; Crews et al., 2003; Devitt and Wilton, 2001).

The present study showed positive phenotypic relationships but negative genetic correlations between serum leptin concentration and daily feed intake, BW and residual feed intake of beef cattle, but not with ADG; the corresponding genetic correlations were negative. The generally low to moderate genetic correlations observed between serum leptin and measures of BW, feed intake, and feed efficiency, despite the moderate to strong genetic correlations may imply correspondingly strong positive environmental correlation of serum leptin with these traits. The phenotypic relationships of serum leptin with DMI and ADG in the present study are similar to the findings of Richardson et al. (2004). These authors observed a significant phenotypic correlation (r = 0.31) between serum leptin concentration and residual feed intake in Angus cattle but no correlations with ADG. Richardson et al. (2004) also observed positive correlations of serum leptin with DMI and feed conversion ratio, though the latter correlations were not statistically significant, possibly due to the limited number of animals used in the study.

A recent study by Brown et al. (2004) in growing Braunvieh-sired crossbred steers (n = 169) and Bonsmara bulls (n = 62) did not show any phenotypic correlations between serum leptin concentration with growth, feed intake or residual feed intake. The positive relationships of serum leptin with BW observed in the present study are consistent with evidence in pigs (Berg et al., 2003) and cattle (León et al., 2004; Liefers et al., 2002). Other evidence in cattle (Garcia et al., 2002) also show that body weight is highly correlated (r = 0.85) to serum leptin concentration. A study in humans (Larsson et al., 1998) showed that subjects with high serum leptin (38.2 ± 8.0 ng/mL) compared to low serum leptin (7.0 ± 1.7) had significantly higher body weight (74.9 ± 8.4 vs. 60.4 ± 5.4 kg), significantly higher body mass index (27.9 ± 2.7 vs. 21.9 ± 2.0 kg m^{-2}), but significantly reduced habitual energy intake (1838 ± 424 vs. 2311 ± 669 kcal/d). The negative genetic correlation between DMI and serum leptin in this study are therefore consistent with the above findings in humans as reported by Larsson et al. (1998).

Kawakita et al. (2001) did not find any relationship between plasma leptin concentration and feed intake in Japanese Black steers. The findings of the present study regarding the phenotypic relationship of serum leptin with feed intake are somewhat in contrast to the negative relationship observed in humans (Larsson et al., 1998). Underestimation of self-reported food intake in humans has been cited as one of the possible reasons that could partially explain the negative correlation between serum leptin and food intake (Larsson et al., 1998). Underestimation of food intake can be about 10% in healthy non-obese men and women and as high as 20% or more in highly obese subjects (Lichtman, 1992). Nevertheless, genetic differences, possibly related to the biological function of leptin in different mammals, may also account for the differences in correlations between circulating leptin levels and voluntary or habitual food intake between humans and cattle.

The results of this study imply that the relationship between serum leptin concentration and body fatness may be stronger than the relationship of serum leptin with feed intake. Delavaud et al. (2000) also observed that the relationship between circulating leptin levels and the day-to-day variation in nutritional status in cattle may be very low (17%) when compared to the relationships of circulating leptin with the long term effect on adipose tissue mass (35%). The molecular mechanisms regulating leptin production remain to be fully understood. However, the stronger relationship between serum leptin and body fatness may partly be due to the fact that several transcription factors that are essential to the differentiation of adipocytes positively regulate the leptin gene promoter (Miller et al., 1996; Mason et al., 1998). Indeed, polymorphisms in the bovine leptin promoter have been shown to have strong associations with serum leptin concentration as well as body fatness (Nkrumah et al., 2005; Liefers et al., 2003).

The weak relationship between serum leptin and feed intake may also be due to the fact that leptin's role in intake regulation is much more central than peripheral, and the saturable transport of leptin into the brain across the blood-brain barrier may be a rate-limiting step with respect to the central role of leptin (Caro et al., 1996). Indeed, doses of leptin that have no impact on food intake regulation when administered peripherally have been shown to considerably reduce food intake when centrally administered (Friedman, 1998). Further studies using relatively larger numbers of animals are required to clearly define both the phenotypic and especially the genetic associations between endocrine leptin and measures of feed intake, BW, growth, feed efficiency and carcass merit of cattle. The availability of molecular data may also be helpful in clearly defining the differences in relationships between circulating leptin and feed intake, growth performance and body composition of different cattle. These results indicated a stronger phenotypic and genetic relationship between serum leptin concentration and measures of ultrasound and carcass merit compared to correlations with performance and feed efficiency. The results of these studies imply that serum leptin concentration can easily be incorporated into appropriate selection programs to favourably improve the carcass merit of cattle.

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Table 6.1. Breed of sire effects (LS means ± SE) on serum leptin concentration, performance, feed efficiency and ultrasound and carcass merits of beef cattle.

	Sire breed				
Traits	Angus	Charolais	Hybrid	SEM	*P* value[a]
Number of animals	205	117	142		
Serum leptin, ng/mL	14.57^{b}	11.59^{c}	14.35^{b}	0.50	<0.001
Average daily gain, kg/d	1.51	1.48	1.46	0.04	0.57
Metabolic size, kg$^{0.75}$	91.25	92.35	90.07	1.25	0.43
Final BW, kg	466.80	468.00	457.70	8.00	0.50
Daily DMI, kg/d	10.69	10.35	10.53	0.20	0.57
Feed conversion, kg DM/kg gain	7.18	7.18	7.40	0.16	0.42
Phenotypic RFI, kg/d	0.06^{b}	-0.25^{c}	0.09^{b}	0.10	0.03
Genetic RFI, kg/d	-0.12^{b}	-0.35^{c}	-0.06^{b}	0.07	0.03
Partial efficiency of growth	0.29	0.29	0.28	0.004	0.43
Ultrasound backfat, mm	10.05^{b}	7.29^{d}	9.29^{b}	0.32	<0.001
Ultrasound LM area, cm^2	82.52^{c}	84.86^{b}	82.97^{c}	0.73	0.09
Ultrasound marbling score	5.39^{b}	4.64^{c}	5.32^{b}	0.10	<0.001
Number of steers	161	109	111	-	-
Carcass weight, kg	315.2	316.6	309.7	5.40	0.56
Carcass grade fat , mm	11.80^{b}	8.14^{c}	11.06^{b}	0.45	<0.001
Carcass LM area, cm^2	82.16^{c}	87.09^{b}	83.11^{c}	1.18	0.03
Carcass marbling score	2.60^{b}	2.22^{c}	2.58^{b}	0.07	0.002
Carcass lean meat yield, %	56.70^{d}	60.12^{b}	57.69^{c}	0.46	<0.001
Carcass quality grade	2.53	2.49	2.48	0.08	0.82
Carcass yield grade	1.88^{b}	1.40^{c}	1.72^{b}	0.07	<0.001

[a] *P* values from overall F test.

[b, c, d] Within a row, means without a common superscript letter differ ($P < 0.05$).

Table 6.2. Relationship of serum leptin concentration (correlations and LS means ± SE) with feed intake and different measures of feed in composite beef cattle.

	Correlation		Serum leptin concentration[a]				
Traits[b]	Phenotypic	Genetic	High	Medium	Low	SEM	*P* value[c]
Animals	464	464	135	172	157	-	-
SLPT	-	-	20.32^{d}	13.78^{e}	8.57^{f}	0.26	<0.001
DMI	0.15	-0.29 ± 0.24	10.96^{d}	10.57^{e}	10.26^{f}	0.15	<0.001
FCR	0.01	-0.44 ± 0.24	7.45	7.28	7.24	0.14	0.48
RFIp	0.04	-0.24 ± 0.38	0.14^{d}	0.02^{de}	-0.15^{e}	0.09	0.037
RFIg	0.03	-0.41 ± 0.25	0.04^{d}	-0.12^{de}	-0.28^{e}	0.09	0.048
PEG	-0.04	0.34 ± 0.24	0.276^{d}	0.287^{e}	0.296^{f}	0.004	0.011

[a] Serum leptin classes are defined as High (> 0.5 SD above the mean), Medium (within ± 0.5 SD of the mean), and Low (< 0.5 SD below the mean);

[b] SLPT = serum leptin concentration (ng/mL); FCR = feed conversion ratio (kg DM/kg gain); RFIp = phenotypic residual feed intake (kg DM/d); RFIg = genetic residual feed intake (kg DM/d); PEG = partial efficiency of growth

[c] *P* values from overall F test.

[d, e, f] Least squares means within rows followed by different superscripts differ ($P < 0.05$).

Table 6.3. Relationship of serum leptin concentration (LS means ± SE) with growth, BW and ultrasound measurements in composite beef cattle.

Traits	Serum leptin concentration[a]			SEM	*P* value[c]
	High	Medium	Low		
Number of animals	135	172	157		
Average daily gain DG, kg/d	1.50	1.48	1.45	0.04	0.49
Metabolic BW, $kg^{0.75}$	92.65[d]	91.26[de]	90.18[e]	0.91	0.02
Final BW[b], kg	477.48[d]	465.69[de]	457.83[e]	5.83	0.002
Slaughter weight, kg	551.48[d]	539.77[de]	528.48[e]	6.74	0.009
Ultrasound backfat, mm	10.40[d]	9.01[e]	7.77[f]	0.29	<0.001
Ultrasound LM area, cm^2	84.24	82.79	83.92	0.78	0.31
Ultrasound Marbling score	5.30[d]	5.11[de]	4.95[e]	0.09	0.001

[a] Serum leptin concentration classes are defined as High (> 0.5 SD above the mean), Medium (within ± 0.5 SD of the mean), and Low (< 0.5 SD below the mean);

[b] Final BW equals weight at end of performance testing.

[c] *P* values from overall F test.

[d, e, f] Least squares means within rows followed by different superscripts differ ($P < 0.05$).

Table 6.4. Heritabilities (along diagonal), Genetic (± SE, below diagonal) and phenotypic (above diagonal) correlations among serum leptin concentration, growth, BW and ultrasound measurements in composite beef cattle.

Traits[a]	SLPT	ADG	MWT	SWT	UBF	ULMA	UMAR
SLPT	**0.34 ± 0.13**	0.08	0.17*	0.10	0.41***	-0.19**	0.25**
ADG	0.05 ± 0.28	**0.59 ± 0.17**	0.52***	0.48***	0.20**	0.16*	0.23**
MWT	-0.41 ± 0.27	0.87 ± 0.12	**0.54 ± 0.16**	0.68	0.32**	0.32**	0.26**
SWT	-0.56 ± 0.21	0.72 ± 0.14	0.99 ± 0.001	**0.50 ± 0.15**	0.26**	0.44***	0.15*
UBF	0.76 ± 0.19	0.54 ± 0.19	0.15 ± 0.24	0.24 ± 0.20	**0.59 ± 0.14**	0.06	0.47***
ULMA	-0.71 ± 0.19	-0.08 ± 0.27	0.31 ± 0.25	0.49 ± 0.21	-0.04 ± 0.28	**0.39 ± 0.13**	0.03
UMAR	0.27 ± 0.22	0.45 ± 0.18	0.29 ± 0.27	0.09 ± 0.22	0.84 ± 0.13	-0.26 ± 0.25	**0.75 ± 0.16**

* ($P < 0.05$); ** ($P < 0.01$); *** ($P < 0.001$).

[a] Abbreviations: SLPT = serum leptin concentration (ng/mL); ADG = average daily gain (kg/d); MWT = metabolic BW ($kg^{0.75}$); SWT = slaughter weight (kg); UBF = ultrasound backfat (mm); ULMA = ultrasound LM area; UMAR = ultrasound marbling score.

Table 6.5. Relationship of serum leptin concentration (LS means ± SE) with carcass traits of composite beef cattle.

	Serum leptin concentration[a]				
Traits	High	Medium	Low	SEM	*P* value[b]
Number of animals	111	128	142	-	-
Carcass weight, kg	315.97	313.51	311.96	4.14	0.68
Grade fat, mm	11.84^{c}	10.71^{d}	8.62^{e}	0.42	<0.001
Carcass LM area, cm^{2}	83.74^{d}	83.25^{d}	85.49^{c}	0.96	0.09
Carcass marbling score	2.65^{c}	2.51^{d}	2.37^{e}	0.06	0.003
Carcass lean meat yield, %	56.68^{e}	57.82^{d}	59.71^{c}	0.39	<0.001
Carcass yield grade	1.91^{c}	1.71^{d}	1.39^{e}	0.08	<0.001
Carcass quality grade	2.68^{c}	2.44^{d}	2.42^{d}	0.06	0.004

[a] Serum leptin classes are defined as High (> 0.5 SD above the mean), Medium (within ± 0.5 SD of the mean) and Low (< 0.5 SD below the mean).

[b] *P* values from overall F test.

[c, d, e] Least squares means within rows followed by different superscripts differ ($P < 0.05$)

Table 6.6. Heritability (along diagonal), Genetic (± SE, below diagonal) and phenotypic (above diagonal) correlations among serum leptin concentration and carcass traits in composite beef cattle

Traits[a]	SLPT	CWT	CGF	CREA	CMAR	LMY	YG
SLPT	**0.34 ± 0.13**	-0.06	0.42***	-0.17*	0.28**	-0.38***	0.32***
CWT	-0.46 ± 0.26	**0.33 ± 0.14**	0.28**	0.43***	0.28**	-0.27**	0.23**
CGF	0.54 ± 0.23	0.53 ± 0.18	**0.51 ± 0.15**	-0.25**	0.50***	-0.90***	0.74***
CREA	-0.75 ± 0.20	0.14 ± 0.29	-0.70 ± 0.20	**0.45 ± 0.15**	-0.18*	0.53***	-0.42***
CMAR	0.76 ± 0.21	-0.32 ± 0.23	0.84 ± 0.15	-0.79 ± 0.18	**0.49 ± 0.16**	-0.53***	0.37***
LMY	-0.59 ± 0.22	0.03 ± 0.25	-0.96 ± 0.03	0.79 ± 0.13	-0.95 ± 0.03	**0.63 ± 0.17**	-0.81***
YG	0.39 ± 0.26	0.31 ± 0.26	0.85 ± 0.09	-0.72 ± 0.16	0.95 ± 0.01	-0.99 ± 0.001	**0.58 ± 0.18**

* ($P > 0.05$); ** ($P < 0.01$); *** ($P < 0.001$).

[a] Abbreviations: SLPT = serum leptin concentration (ng/mL); CWT = carcass weight (kg); CGF = carcass grade fat (mm); CLMA = carcass LM area (cm^2); CMAR = carcass marbling [illegible] LMY = lean meat yield (%); YG = carcass yield grade.

CHAPTER 7

Single Nucleotide Polymorphisms in the Bovine Leptin Gene and their Associations with Growth, Feed Intake, Feed Efficiency, and Carcass Merit of Composite Cattle[3]

7.1 Introduction

Leptin is an adipocyte-derived 16-kDa cytokine-like hormone product of the obese gene (Zhang et al., 1994; Ji et al., 1998) that circulates in the serum in the free and bound forms. Leptin's role as a lipostatic signal regulating whole body energy metabolism through interactions with the leptin receptor in the hypothalamus makes it one of the best physiological markers for the regulation of BW, feed intake, energy expenditure, body fatness (Houseknecht et al., 1998; Woods et al., 1998), reproduction (Cunningham et al., 1999; Garcia et al., 2002), and certain immune system functions (Lord et al., 1998). Circulating leptin or tissue mRNA levels are correlated with body weight, food intake, and body fatness in humans or animals (Larsson et al., 1998; Delavaud et al., 2002; Berg et al., 2003). In cattle, circulating leptin levels are correlated with the regional distribution of body fat (Yamada et al., 2003) and carcass merit (Minton et al., 1998; Geary et al., 2003).

The leptin gene was mapped to bovine chromosome 4 (Stone et al., 1996). Konfortov et al. (1999) sequenced the leptin gene across a diverse panel of cattle and identified a total of 20 exonic single nucleotide polymorphisms (**SNP**) at a frequency of one per 84 base pairs (**bp**). Subsequently, several studies involving these SNP have been performed to detect associations with serum leptin concentration (Liefers et al., 2003), feed intake (Lagonigro et al., 2003; Liefers et al., 2002; Oprzadek et al., 2003), milk yield (Liefers et al., 2002; Buchanan et al., 2003), and body fatness (Buchanan et al., 2002; Nkrumah et al., 2004a; Nkrumah et al., 2005; Schenkel et al., 2005; Woodward et al., 2005).

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[3] Revised sections of this chapter have been published; Nkrumah et al., 2004, Can. J. Anim. Sci. 84: 211–219; Nkrumah et al., 2005; J. Anim. Sci. 83:20–28

On the other hand, the leptin promoter regulates the expression of the leptin gene and polymorphisms in this region are generally associated with the sequence elements and factors regulating gene expression and may completely abolish the inducibility of the promoter or reduce its activity significantly. Several potential transcription factor binding sites have been detected in the leptin promoter region of several mammalian species including the CAAT/enhancer binding proteins (C/EBP, Miller et al., 1996; Taniguchi et al., 2002), peroxisomal proliferator-activated receptor receptor γ (PPAR-γ, Hollenberg et al., 1997), SP1 and LP1 (Fukuda and Iritani, 1999; Mason et al., 1998), and HIF-1 (Meissner et al., 2003).

The bovine leptin promoter has been sequenced (GenBank accession no. AB070368) (Taniguchi et al., 2002). Several polymorphisms have been detected in the leptin promoter and associations with serum leptin concentration, performance, and carcass merit have been reported (Liefers et al., 2005; Nkrumah et al., 2005; Schenkel et al., 2005). The objective of this study was to examine associations of polymorphisms in the leptin gene and leptin promoter with serum leptin concentration, growth, BW, feed intake, feed efficiency, feeding behaviour, and carcass merit.

7.2 Materials and Methods

7.2.1 Animals and Phenotypic Data

Growth, feed intake, feeding behaviour, temperament, ultrasound and carcass data were collected on 464 composite beef steers sired by Angus, Charolais or University of Alberta Hybrid bulls between 2002 and 2005. The Dams used were produced from crosses among three composite cattle lines, namely Beef Synthetic 1 (BS1), Beef Synthetic 2 (BS2) and Dairy X Beef Synthetic (DBS) (Goonewardene et al., 2003). Cows and heifers for the study were bred in multiple sire breeding groups on pasture and the sire of each calf was later determined in a parentage test using a panel of bovine microsatellite markers.

The animals were managed and tested under feedlot conditions using the GrowSafe automated feeding system (GrowSafe Systems Ltd., Airdrie, Alberta, Canada) at the University of Alberta's Kinsella Research Station. Details of the procedures for the feedlot tests were given by Nkrumah et al. (2004b) and are as detailed out in chapter three. The animals weighed 353.0 (SD = 61.3) kg and were 252 (SD = 42) days of age at the beginning of tests. Two tests made up of approximately 80 animals per test were conducted each year. The animals used in the study were cared for according to the guidelines of the Canadian Council on Animal Care (CCAC, 1993). In year one, steers were fed free choice a backgrounding diet of mainly alfalfa-brome hay with oats supplemented with corn grain and feedlot mineral supplement to promote a growth rate of just under 1.0 kg/d for approximately 30 d. This period was followed by a 30-d pre-test adjustment period in which the amount of corn in the backgrounding diet was adjusted up gradually to introduce the animals to the test diet and the feeding system. This was done to allow them to adapt to the diet and learn to feed from the test facility.

The test diet in year one was composed 80.0% dry-rolled corn, 13.5% alfalfa hay pellet, 5% feedlot supplement (32% CP beef mineral supplement) and 1.5% canola oil, supplying approximately 2.90 Mcal/kg of ME and 12.5% CP. In yr two and three, the same test procedures were used, but the test diet contained 64.5% barley grain, 20% oat grain, 9.0% alfalfa hay pellet, 5.0% beef feedlot supplement and 1.5% canola oil, supplying 14.0% CP and 2.91 Mcal/kg of ME. Corn was used in yr 1, instead of barley and oat, because of a feed barley shortage that particular yr.

7.2.2 *Blood collection and serum leptin assay*

A 10-mL blood sample was collected by venipuncture from each animal at the start of the feed efficiency tests from which genomic DNA was extracted using a standard saturated salt phenol/chloroform procedure (Miller et al., 1988). At the end of the feed efficiency tests (one week prior to slaughter), blood samples were collected from each animal by jugular venipuncture into Vacutainer tubes. Animals were bled in the morning and all animals were allowed unrestricted access to feed and water prior to bleeding. Blood samples were allowed to clot for approximately 18 h at 4°C. Samples were centrifuged at 2,500g for 30 minutes, and serum collected and stored at -20°C until assayed for leptin using the leptin radio-immuno assay (RIA) kit described by Delavaud et al. (2000). Intra- and interassay coefficients of variation for the leptin assays were less than 5%.

7.2.3 Traits Analyzed and their Derivations

Traits analyzed include serum leptin concentration, growth, feed intake, feed efficiency, feeding behaviour, ultrasound and carcass merit. Procedures for obtaining the measures of feedlot performance and feed efficiency have been described previously (Nkrumah et al., 2004b). Linear regression in SAS (SAS Institute, Inc., Cary, NC, version 9.1.3) of weekly or fortnightly BW against time (d) was used to derive ADG, final BW and mid-test metabolic size (**MWT**, $BW^{0.75}$) for each steer. The total feed intake of each animal over a 70-d test period was used to compute the daily DMI. Feed conversion ratio (**FCR)** was computed as the ratio of DMI to ADG on test. The partial efficiency of growth (PEG; i.e., energetic efficiency for ADG above maintenance) of each animal was computed as the ratio of ADG to the difference between average daily DMI and expected DMI for maintenance (**DMIm**) (Arthur et al., 2001), where DMIm was computed according to NRC (1996).

Residual feed intake (**RFI**, an alternative measure of feed efficiency) was calculated both from phenotypic regression (**RFIp,** Arthur et al., 2001) or genetic regression (**RFIg**, Crews et al., 2005; Hoque et. al., 2005) of ADG and MWT on DMI. Test group (six levels) was included as an independent variable in the calculation of RFI. In each case, individual RFI was computed as actual DMI minus the expected DMI predicted from the appropriate phenotypic or genetic regression model. Ultrasound backfat thickness (**UBF**), LM area (**ULMA**), and marbling score (**UMAR**) were predicted from linear regressions against time of measurements obtained every 28 days with an Aloka 500V real-time ultrasound with a 17 cm, 3.5-MHz linear array transducer (Overseas Monitor Corporation Ltd., Richmond, BC).

The feedlot behaviour traits studied were daily feeding duration (**FD**), daily feeding "head down" time (**HD)**, daily feeding frequency (**FF,** i.e. feeding frequency) and flight speed (**FS,** as a measure of temperament, Burrow et al., 1988). Procedures for determining the feeding behaviour traits from the GrowSafe System have previously been described (Basarab et al., 2003). Daily feeding duration was computed as the sum of the difference between feeding event end-times and start-times per day for each animal. It was equal to the total number of minutes each day spent in feeding-related activities (prehension, chewing, backing away from the bunk and chewing, socializing, scratching, or licking) at the feedbunk. Head down time refers to the sum of the number of times the animal's electronic identification (transponder) was detected by the GrowSafe System during a feeding event multiplied by the system's scanning time, where scanning time is system dependent and ranges from 1.0 to 6.3 s. Daily feeding frequency in this study was defined as the number of independent feeding events for a particular animal in a day. A feeding event starts when an animal's transponder is first detected and ends when the time between the last two transponder readings on that animal was greater than 300 s or when a different transponder number is encountered. Exit velocity was measured as the rate (m/s) the animals exited a squeeze chute and traversed a fixed distance of 8 feet, with infrared sensors used to trigger the start and stop of the timing system.

At the end of the feed efficiency tests animals were weighed and shipped to a commercial packing plant where they were harvested the following day and standard industry carcass data collected after a 24-h chill at -4°C. Carcass traits were evaluated according to the Canadian beef carcass grading system (Agriculture Canada, 1992). Carcass weight (**CWT**) of each animal was determined as the weight of the left and right halves of the carcass after 24-h chill at -4°C. Carcass grade fat (**CGF**) was measured at the 12/13th rib of each carcass. Carcass marbling score (**CMAR**) is a measure of intramuscular fat and can be classified as 1 to < 2 units = trace marbling (Canada A quality grade, **QG**); 2 to < 3 units = slight marbling (Canada AA quality grade); 3 to < 4 units = small to moderate marbling (Canada AAA **QG**) and ≥ 4 units = slightly abundant or more marbling (Canada Prime). Lean meat yield (**LMY**) is an estimate of saleable meat and was calculated according to the equation, lean % = 63.65 + 1.05 (muscle score) - 0.76 (grade fat). Yield grade (**YG**) is the proportion of lean meat and is classified as Y1 = >59 %, Y2 = 54 to 58%, and Y3 < 54%.

7.2.4 SNP Identification and Genotyping

Table 7.1 presents the general information on all the SNP considered in the present study. The identification of UASMS2 and UASMS3 in the bovine leptin promoter utilized the approximately 3.0 kb 5' flanking region upstream from the putative transcription start site as reported by Taniguchi et al. (2002) (GenBank accession no. AB070368). Sixteen animals with extreme phenotypes for feed intake and backfat thickness were selected from the population of animals with data. Primers were designed to cover the entire promoter region, and genomic DNA from the panel of 16 selected animals was amplified by polymerase chain reaction (PCR) in two steps. A single PCR reaction (50 µL) of the first amplification step contained 1X high fidelity PCR buffer, 250 µM dNTPs, 2 mM $MgSO_4$, 15 pmoles of forward and reverse primers, 1U Platinum high fidelity *Taq* DNA Polymerase (Invitrogen, Burlington, Ontario), and 20 to 60ng of genomic DNA. Amplification was carried out on a GeneAmp 9700 (Applied Biosystems, Streetsville, Ontario) with the following conditions: 2 min at 94°C, 5 cycles of 94°C for 60 s, 55°C for 30 s, 68°C 30 s followed by 30 cycles of 94°C, 55°C, 68°C at 30 s each and a final step of 68°C for 5 min.

The second amplification step used the same conditions as the first except that nested forward and reverse primers were used, and the template was 2 µL of PCR product from the first amplification. In addition, the second thermal cycling step of 5 cycles at 94°C for 60 s, 55°C for 30 s and 68°C for 30 s was also excluded. The PCR products were purified for sequencing by digesting with ExoI and shrimp alkaline phosphatase (SAP) at 37°C for 15 minutes, and the enzymes deactivated at 85°C for 15 minutes. The PCR products from each animal were used for direct sequencing on a Beckman CEQ 8000 Genetic Analysis System (Beckman Coulter Canada Inc., Mississauga, Ontario). Forward and reverse sequences from each animal were comparatively aligned and analyzed for polymorphisms using the Discovery Studio Gene (DS Gene) v 1.1 DNA sequence analyses software (Accelrys Inc., San Diego, CA, USA).

Respectively, UASMS2 (C-T substitution) and UASMS3 (G-C substitution) are located at nucleotide positions 528 and 1759 in the leptin promoter (GenBank accession no. AB070368) (Nkrumah et al., 2005). A third SNP, a C/T substitution at the 207 nucleotide position in the leptin promoter was found to be in complete linkage disequilibrium (**LD**) with UASMS3 and was therefore not analyzed further. The E2JW SNP is an A-T mutation at position 252 in exon 2 of the leptin gene that results in a non-conservative amino acid substitution, from tyrosine (TAT) to phenylalanine (TTT), (GenBank accession no. AY138588). It has previously been shown to be associated with feed intake (Lagonigro et al., 2003). The R4C (E2FB) SNP is a C-T missense mutation at position 305 in exon 2 of the leptin gene (GenBank accession no. AY138588; position 466 in EMBL Accession no. AJ512638). The R4C SNP results in an arginine (R; CGC) to Cystein (C; TGC) substitution at amino acid 4 (R4C) in the leptin molecule. This confers a putative conformational change that has been linked with variations in carcass fatness in beef cattle (Buchanan et al., 2002) as well as milk and protein yield in dairy cattle (Buchanan et al., 2003). The E2SL SNP is an A-G substitution at position 600 in exon 2 of the leptin gene (GenBank accession no. BTA236854). The A59V mutation is a C-T SNP at position 321 (GenBank accession no. BTA512639; EMBL Accession no. AJ512639). The SNP results in an alanine (A; GCG) to valine (V; GTG) at amino acid 59 in the β-helix region that is conserved between species in the leptin molecule. The A59V SNP has previously been shown to be associated with serum leptin concentration in dairy cows (Liefers et al., 2003). The E3SL is a C-T mutation at position 396 in exon 3 of the leptin gene (GenBank accession no. BTA132764).

Initially, the R4C SNP was genotyped in a total of 144 animals from five commercial lines of relatively unrelated cattle (selection lines M1, M2, M3, M4, and TX of Beefbooster Inc., Calgary, Canada). The initial genotyping of UASMS2, UASMS3, E2JW, and R4C SNP was carried out using the 5' nuclease allelic discrimination assay on an ABI PRISM 7700 sequence detector (Applied Biosystems, Streetsville, Ontario). Forward and reverse primers and two ABI TaqMan fluorogenic probes (with a different reporter dye on each probe to target the two alleles of each SNP) were designed to amplify each polymorphism using genomic DNA from each animal. A perfect match of a probe sequence to the target sequence will result in amplification during which cleavage and release of the reporter dye occurs.

A substantial increase in fluorescence signal for one or the other of the two dyes indicates homozygosity for a particular allele of the SNP whereas an increase in both dyes represents heterozygosity. A subset of the genotyped animals was sequenced across each polymorphism and the sequence results were used to confirm the genotypes obtained by discrimination assays. Subsequent genotyping of all SNP for the 464 animals used in the study was carried out using the Illumina GoldenGate assay on the BeadStation 500G Genotyping System (Illumina Inc., San Diego, CA). The GoldenGate assay uses an allele-specific extension reaction and universal PCR technology to multiplex simultaneously and genotype up to 1,536 SNP loci from approximately 250 ng genomic DNA (Oliphant et al., 2002; Shen et al., 2005).

7.2.5 *Statistical Analyses*

χ^2 tests to examine the genotype frequencies of each polymorphism for deviations from Hardy-Weinberg equilibrium (**HWE**) and tests of allelic associations between each SNP pair to detect the degree of LD were performed using PROC ALLELE of SAS/Genetics 9.1.3 (SAS Institute, Inc., Cary, NC). SAS PROC ALLELE calculates the allele and genotype frequencies, polymorphism information content (PIC), heterozygosity, and other measures of marker informativeness for each marker using maximum likelihood estimation. Estimates of p–values and standard errors were obtained by permutation and bootstrapping, respectively.

The R4C SNP was initially tested for association with feed intake, feed efficiency, growth, real-time ultrasound measurements, feeding behaviour, carcass traits, and body composition using data on a total of 144 from each of the five genetic selection lines (M1, M2, M3, M4, and TX) of a commercial population of beef cattle (Nkrumah et al., 2004a). Foundation breeds were Angus for M1, Hereford for M2, various small breeds for M3, Limousin and Gelbvieh for M4, and Charolais for TX. The effect of different R4C genotypes were tested by least squares procedures using PROC GLM of SAS. The model included fixed effect of SNP genotype (CC, CT and TT), fixed effect of genetic selection line (M1, M2, M3, M4 and TX), year of test (two levels), herd of origin nested within selection line and year, possible interactions between fixed effects, and linear covariates of age of animal, days on test, and age of dam. The initial statistical model included the effect of age of dam, but dam age was excluded from the final model, as it had no significant effect on any of the traits analyzed (*P* > 0.10). Multiple comparisons of trait means for SNP genotypes were analyzed by least squares.

Using data on the 464 animals genotyped in the experimental population, single marker association analyses were carried out to evaluate the relationship between different genotypes of each SNP and serum leptin concentration, growth rate, BW, feed intake, feed efficiency, feeding behaviour, and ultrasound and carcass merit. Effects of SNP genotype were analyzed by least squares procedures using PROC MIXED of SAS (SAS Institute, Inc., Cary, NC, version 9.1.3). The statistical model used included fixed effects due to SNP genotype (two or three levels depending on SNP), breed of sire (Angus, Charolais, and Hybrid), year of test (three levels), test group nested within year (two levels per year), all possible interactions, and linear and quadratic effects of age when ($P < 0.05$). All interaction terms that did not account for a significant portion of the observed variance ($P > 0.10$) were subsequently excluded from the final model. Additive effects and dominance deviation were estimated for traits that were different ($P < 0.05$) among different SNP genotypes. Additive genetic effects (***a***) were computed as the difference between the solutions of the estimate for the trait value of the two homozygous genotypes; dominance deviation (***d***) was computed by subtracting the average of solutions of the estimate for the trait value for homozygous genotypes from that of the heterozygote genotype (Falconer and McKay, 1996).

7.3 Results and Discussion

There is considerable interest in application of molecular technologies in the form of specific DNA markers that are associated with various quantitative traits in marker-assistasted selection. This process would promote more efficient and relatively easy selection and breeding of farm animals with an advantage for an inheritable trait of growth rate, BW, carcass merit, feed intake, and milk yield and composition (Spelman and Bovenhuis, 1998). Several candidate genes have been identified in farm animals for association studies based on a known relationship with physiological or biochemical mechanisms involved in the regulation of the trait in question. It is seldom reported that one particular polymorphism in a candidate gene would influence several traits of economic importance in livestock at the same time. However, the leptin gene seems to be one of the exceptions as its involvement in the regulation of several biologically important processes in the body makes it, perhaps, one of the best physiological gauges for energy balance, BW and body composition in mammals. The present study looked at the associations of several polymorphisms in the bovine leptin gene with several traits of economic importance in cattle.

7.3.1 *Allele and genotype frequencies and their relationships*

The C and T alleles of the R4C SNP occurred at frequencies of 0.48 and 0.52, respectively, in the commercial population. Frequencies of the three genotypes of R4C were approximately distributed according to Hardy-Weinberg proportions in the commercial selection lines used in the study ($P > 0.10$, data not shown). The PIC, heterozygosity, and allele frequencies of the different SNP tested in the experimental population are presented in Table 7.2. With the exception of E2SL, the PIC and heterozygosities of all the SNP examined were moderate. The frequency of the C allele of UASMS2, UASMS3, R4C, A59V, and E3SL were 82%, 40%, 42%, 26%, and 24%, respectively. The frequency of the A allele of E2JW and E2SL, respectively, were 26% and 96%. Buchanan et al. (2002), Liefers et al. (2003), Lagonigro et al. (2003), and Schenkel et al. (2005), respectively, reported the frequency of the C allele for R4C to be 54%, 67%, 65%, and 61%. For UASMS2 and UASMS3, the frequency of the C allele observed in the study are consistent with those reported by Crews et al. (2004) and Schenkel et al. (2005). Lagonigro et al. (2003) and Liefers et al. (2003), respectively, reported the frequency of the C allele of A59V to be 54% and 75%. For E2JW, Lagonigro et al. (2003) and Schenkel et al. (2005), respectively, reported the frequency of the A allele to be 86% and 96%; Liefers et al. (2003) did not find this polymorphism after sequencing 60 Holstein-Friesian cows for exons 2 and 3 of the leptin gene.

Table 7.3 presents the χ^2 test statistics and the associated significance levels for the tests of HWE and LD (Falconer and Mackay, 1996) for the various SNP. With the exception of E2JW and E3SL, the observed genotype frequencies of all the SNP agreed with HWE expectations ($P > 0.10$). For E2JW, the A allele was observed more than expected according to HWE proportions ($P < 0.001$). The T allele of E3SL was observed more than expected according to HWE proportions ($P < 0.05$). These results are consistent with the findings of Buchanan et al. (2002), Lagonigro et al. (2003), and Schenkel et al. (2004). Pairwise comparison of genotypes revealed the existence of significant linkage disequilibrium between most of the SNP considered in the study ($P < 0.001$). This finding is not surprising due to the fact that all the SNP exmined are located so close together along the leptin gene. The UASMS2 and E2JW SNP were jointly in linkage equilibrium ($P > 0.10$), which is consistent with the finding of Schenkel et al., 2005). Joint linkage equilibrium was also detected between E2JW with E2SL and E3SL and between E2SL with A59V and E3SL ($P > 0.10$).

7.3.2 *SNPs Dropped from the Final Analyses*

The number of animals with records, mean, and SD, of the traits considered in the study are shown in Tables 3.2 and 7.4. Single marker associations with the data from the commercial population, (Table 7.4, n = 144) were carried out only for R4C and E2JW. All other associations were conducted with the experimental population dataset (with known pedigree information; Table 3.2). Of the SNP considered, E2JW, and E2SL were not associated significantly with any of the test traits ($P > 0.10$). The measures of LD between E2SL with the other SNP were the lowest of all the SNP considered, thus it was not surprising that this polymorphism was unrelated to any of the test traits. No report was found in the literature considering associations of E2SL with any traits in beef or dairy cattle. With respect to E2JW, the findings of this study are in contrast to that reported by Lagonigro et al. (2003), who showed that animals with the AT genotype had significantly higher feed intake compared to individuals with AA genotype. In addition, these results contrast the findings of Schenkel et al. (2005), who observed significant associations of E2JW with fat yield, lean yield, and grade fat.

The E3SL SNP was significantly associated with serum leptin concentration ($P < 0.001$); the difference in serum leptin concentration between CC and TT animals was 5.80 ng/mL. However, despite this strong association with serum leptin concentration, E3SL was not associated with any other test trait ($P > 0.10$). The strong associations of E3SL with serum leptin concentration may be attributable to its strong LD with other SNP that show associations with serum leptin concentration. Association of the UASMS3 in the leptin promoter with other traits was initially tested (Nkrumah et al., 2005) with the year one data of the experimental population (n = 150). The results indicated associations of the GG genotype of UASMS3 with increased BW ($P < 0.05$), DMI ($P < 0.01$), daily feeding duration ($P < 0.01$), and ultrasound backfat thickness ($P < 0.05$); there were also trends for associations with ADG, feeding frequency, and phenotypic RFI ($P < 0.10$). Schenkel et al. (2005) reported an association of UASMS3 with fat yield but not with other carcass traits. Additionally, Woodward et al. (2005) reported associations of this SNP with BW, carcass weight, and carcass backfat. Differences between genotypes of UASMS3 in the subsequent analyses carried out on the complete dataset (n = 464, Table 3.1) followed similar trends as observed in the year one dataset; however, none of the associations were statistically significant (P > 0.10).

7.3.3 Associations with R4C

Associations involving the R4C SNP were carried out first in the commercial population (Nkrumah et al., 2004a), and subsequently in the experimental population. Associations of R4C with various commercial population test traits are presented in Table 7.5. In the commercial population, steers carrying the T allele of the R4C had a higher rate of gain in ultrasound backfat (*P* = 0.02), tended to have higher ultrasound backfat (*P* = 0.07), had higher carcass grade fat (*P* < 0.01), lower yield grade (*P* < 0.01), and lower lean meat yield (*P* < 0.01). No associations were found among the different R4C genotypes and real-time ultrasound marbling, ultrasound LM area, carcass marbling, carcass LM area or any other test trait (*P* > 0.10). In addition, no associations were found for growth rate, feed intake, feed conversion ratio, or phenotypic RFI. Estimated additive genetic effects for R4C genotypes were 0.04 mm/d, 1.46 mm, 2.82 mm, 0.48 units, and 2.34% for gain in ultrasound backfat, final ultrasound backfat, carcass grade fat, yield grade, and lean meat yield, respectively.

In the experimental population (Table 7.6), R4C was associated with ultrasound backfat (*P* = 0.059), tended to be associated with carcass grade fat and average carcass backfat (*P* < 0.10), was associated with carcass LM area (*P* = 0.026), and tended to be associated with carcass lean meat yield (*P* = 0.08). In addition, flight speed was significantly higher (*P* < 0.01) in animals with CC genotype of R4C compared to animals with CT or TT; the difference between CC and TT genotypes in flight speed was 0.36 m/s Additive genetic effects for ultrasound backfat, grade fat, carcass LM area and lean yield, respectively, were 0.98 mm, 1.20 mm, 3.26 cm^2, and 1.29%.

However, the R4C polymorphism was not associated with serum leptin concentration (*P* = 0.97), ultrasound marbling (*P* = 0.97), or carcass marbling (*P* = 0.63). No associations were found for growth, BW, feed intake, phenotypic RFI, genetic RFI, feed conversion ratio, or the remaining test traits in the study (*P* > 0.10). The associations between R4C and carcass quality traits observed in the present study are consistent with the findings of Buchanan et al. (2002) and Schenkel et al. (2005). In addition, Woodward et al. (2005) reported associations between this SNP and LM area, carcass backfat, and carcass yield grade. However, Crews et al. (2004) using data on 433 Charolais and Charolais-cross steers did not find association of R4C with backfat, carcass marbling or any other carcass trait. Crews et al. (2004) also did not find any associations with any of the performance or feed efficiency traits considered in their study. Though Buchanan et al. (2003) reported associations between R4C and milk and protein yield, Liefers et al. (2003) did not find any associations between this mutation and milk yield or associated traits. Generally, backfat thickness may be positively correlated genetically and phenotypically with marbling score, especially in older animals. Thus, many attempts have been made to extrapolate and even equate the associations of the R4C SNP in some studies to an association with marbling score. However, in the present study, no associations were observed between R4C and marbling score, despite strong genetic (r = 0.84 ± 0.13) and phenotypic (r = 0.47) correlations between backfat and marbling score.

The lack of association between R4C and serum leptin concentration observed in this study is in contrast to the findings of Liefers et al. (2003) who reported associations of the CC genotype of R4C with significantly higher serum leptin concentration during late pregnancy and first few days in lactation in dairy cows. Higher circulating leptin in CC animals is somewhat surprising since all reported studies (even without significant associations) indicate CC animals to have lower body fatness and higher leanness. In addition, it is illogical that CC animals that exhibit significantly lower leptin mRNA levels (Buchanan et al., 2002) will subsequently have higher circulating leptin levels. It is possible that the associations of R4C with circulating leptin observed by Liefers et al. (2003) may be pregnancy-induced or may be the result of LD with one of the polymorphisms in the leptin gene or promoter. These results, as well as results from other studies, show that animals carrying the T allele of R4C versus the C allele may produce carcasses with higher subcutaneous fat but lower lean yield and yield grades, but do not differ in carcass marbling or other fat depots.

7.3.4 *Associations with UASMS2*

Associations of UASMS2 with performance, feed efficiency, feeding behaviour, and carcass merit were initially considered in the yr 1 dataset of the experimental animals (Nkrumah et al., 2005; Table 7.7). Of the 150 animals in the analysis, there were 99, 45, and 6 animals with genotypes CC, CT, and TT, respectively. Animals with the TT genotype had significantly higher serum leptin concentration (a = 11.24, d = 4.49 ng/mL; $P < 0.001$). In addition, animals with the TT genotype had significantly higher ADG ($P = 0.002$; a = 0.10 kg/d), metabolic BW ($P = 0.05$; a = 5.94 $\text{kg}^{0.75}$), daily DMI ($P = 0.001$; d = 0.59 kg/d), and daily feeding duration ($P = 0.02$; d = 5.31 min/d). Also, phenotypic RFI and final BW tended to differ among animals with different genotypes ($P < 0.10$). In addition, the TT genotype was significantly associated with higher ultrasound backfat ($P = 0.001$; a = 3.58 mm) and ultrasound marbling score ($P = 0.01$; a = 0.68, d = 0.34).

Table 7.8 shows the associations of UASMS2 with different traits using the complete dataset from the experimental population. Of the 464 animals in the complete dataset, there were 306, 146, and 12 animals with genotype CC, CT, and TT, respectively. Animals with the TT genotype had significantly higher serum leptin concentration ($P <$ 0.001; a = 6.40, d = 2.29 ng/mL), daily DMI ($P = 0.036$; a = 0.76 kg/d), daily gain in ultrasound backfat ($P = 0.017$), gain in ultrasound marbling ($P = 0.05$), final ultrasound backfat ($P = 0.018$; a = 2.43, d = 1.11 mm), and final ultrasound marbling score ($P = 0.02$; a = 0.51). Genetic and phenotypic RFI tended to be higher ($P < 0.10$) in TT animals compared to CC animals. The associations with growth rate, BW, and feeding duration observed in the yr 1 dataset were not observed ($P > 0.10$) following analysis of the complete dataset, though the direction of differences were consistent. Of the 381 animals with carcass data, there were 255, 118, and 8 animals with genotypes CC, CT, and TT, respectively. Carcass grade fat (a = 3.04 mm) and average carcass backfat (a = 2.99 mm) tended to be higher ($P < 0.10$) in TT animals compared to CC animals.

Though CC animals had 2.40% higher carcass lean meat yield compared to TT animals, this difference was not statistically significant ($P > 0.10$). Association of UASMS2 with the remaining test traits were not significant ($P > 0.10$). In a group of 433 Charolais and Charolais-cross steers, Crews et al. (2004) reported associations of this SNP with daily DMI, RFI, carcass weight, carcass marbling score, and LM area, but not with ADG or carcass grade fat. Woodward et al. (2005) reported associations of this SNP with carcass weight, LM area, and dressing percentage. Contrary to these findings, Schenkel et al. (2005) did not find any associations of this SNP with a variety of carcass and meat quality traits in a population of 1,111 crossbred bulls, heifers, and steers.

7.3.5 Associations with A59V

Associations of A59V with various traits are indicated in Table 7.9. Of the 464 animals with total phenotypic records, 31, 174, and 259, respectively, had genotypes CC, CT, and TT. The TT genotype of A59V was significantly associated with higher serum leptin concentration ($P = 0.003$; $a = 3.89$ ng/mL), ADG ($P = 0.04$; $a = 0.13$, $d = -0.06$ kg/d), and FCR ($P = 0.005$; a = -0.68, $d = 0.25$ kg DM/kg gain). There were also associations with other measures of feed efficiency including relative growth rate and Kleiber ratio ($P = 0.013$) and a trend towards an association with PEG ($P < 0.06$). The A59V SNP was also associated with ultrasound backfat thickness ($P = 0.014$; $a = 1.53$ mm) and ultrasound LM area ($P = 0.011$; $d =1.89$ cm^2) but not with ultrasound marbling score ($P > 0.10$). There were no significant associations with BW, daily DMI, phenotypic or genetic RFI (P > 0.10). Of the 381 animals with carcass data available, 26 were CC, 143 had CT genotype and 212 had TT genotype. Differences were observed among genotypes in carcass grade fat (P = 0.10), average carcass backfat ($P = 0.039$; $a = 1.92$ mm), carcass LM area ($P = 0.015$; $a = -1.57$, $d = -1.72$ cm^2), carcass lean meat yield ($P = 0.024$; $a = 1.66\%$), and yield grade ($P = 0.10$).

The association of A59V with serum leptin concentration is consistent with the findings of Liefers et al. (2003) who reported higher serum leptin levels in TT animals during late pregnancy, but not in lactation. No other association study has been carried out with this particular mutation, and Liefers et al. (2003) attributed the observed associations of A59V with serum leptin concentration to the strong LD it shares with the R4C SNP. However, as stated previously, it is unlikely for the R4C SNP to be the causative mutation for increased leptin levels. This is because it is the TT genotype that has been shown to be associated with increased fatness and leptin mRNA levels (Buchanan et al., 2002; Nkrumah et al., 2004a; Schenkel et al., 2005; Woodward et al., 2005).

7.3.6 Associations with UASMS2-A59V Genotype Combinations

Due to the strong associations observed individually between UASMS2 and A59V with serum leptin concentration, growth rate, feed efficiency and carcass quality, further analyses of the two SNP were carried out to test for effects of their genotype combinations resulting from the significant LD between the two SNP (Table 7.10). Of the 464 animals with data for all traits, 31, 127, 148, 47, 99, and 12 had with genotype combinations CC-CC, CC-CT, CC-TT, CT-CT CT-TT, and TT-TT, respectively (counting UASMS2 genotypes first). The analysis showed that the T alleles of both SNP show associations with higher serum leptin concentration, with the highest effect observed for genotype TT-TT (*P* < 0.01). Regression analysis using dummy variables revealed that differences in genotype combinations accounted for 9.0% of the variation in serum leptin concentration.

There were significant differences among the different genotype combinations on ADG (*P* = 0.04), FCR (*P* = 0.019), relative growth rate (*P* = 0.004) Kleiber ratio (*P* = 0.005), with the highest effects on growth and feed efficiency clearly attributable to the presence of two copies of the T allele of A59V. On the other hand, the T alleles of both SNP were associated with increased ultrasound backfat (*P* = 0.005) and marbling score (*P* < 0.001). The presence of two copies of the T allele of UASMS2 was associated with the highest effects on ultrasound marbling and backfat thickness. The differences in genotype combinations accounted for 6.68% and 6.26% of the variation in ultrasound backfat and marbling score, respectively. The presence of the CT genotype of A59V only or CTCT genotype of both SNP was associated with higher ultrasound LM area (*P* = 0.01). However, no significant differences were observed among the different genotype combinations in metabolic BW, daily DMI, PEG, genetic, and phenotypic RFI (*P* > 0.10).

Of the 381 animals with carcass data, there were 26, 109, 120, 34, 84, and 8 animals with genotype combinations CC-CC, CC-CT, CC-TT, CT-CT CT-TT, and TT-TT, respectively (counting UASMS2 genotypes first). The T alleles of both SNP were associated with increased carcass fatness, with the TT-TT genotype combination showing the strongest effect on increased carcass backfat and grade fat (*P* < 0.001). Differences in genotype combinations accounted for approximately 6.0% of carcass fatness. The T alleles of both SNP were generally associated with increased carcass marbling (4.2% of variation in marbling score), with the TT-TT genotype combination showing the highest carcass marbling score ($P < 0.001$). On the other hand, the C alleles of both SNP show associations with increased lean yield ($P < 0.001$) (accounting for 8.92% of carcass lean meat yield). The differences in carcass LM area were consistent with that of ultrasound LM area. The presence of the CT genotype of A59V only, or CTCT genotype of both SNP, was associated with higher carcass LM area ($P < 0.001$); differences in genotype combination accounted for 4.85% of the variation in carcass LM area.

In summary, not all of the statistically significant associations observed in the analysis may be biologically or economically relevant. The exact molecular and physiological mechanisms underlying the associations of the polymorphisms with the variety of traits reported in the present study are currently unknown. The strong interest in the R4C mutation is due to the addition of an unpaired cystein to the leptin protein and the associated putative conformation alteration of the tertiary structure of the leptin protein, which has implications for the potential binding of leptin to its receptor (Buchanan et al., 2002). However, in the present study, it is the promoter mutation (UASMS2) and the exon 3 mutation (A59V) that showed the strongest associations (at least statistically) with serum leptin concentration, performance, feed conversion, and carcass merit. The interest in the promoter mutation (UASMS2) lies in its physical proximity to potential transcription factor binding sites and regulatory sequences in the leptin gene. Several putative Sp1, CCAAT/enhancer binding protein (C/EBP), and TATA box binding sequences were detected in the vicinity of the SNP reported in the present study (Taniguchi et al., 2002).

Thus, the association of UASMS2 with serum leptin concentration as well as body fatness provides indirect *in vivo* evidence on the potential biological role of the mutation. Its location in the regulatory region of the leptin gene makes it a potential regulator of leptin expression in cattle or serve as surrogates for causative SNP that are in LD and yet to be established. Studies in humans have shown that mutations in the CCAAT/enhancer binding protein (C/EBP-α) region of the leptin promoter abolished the inducibility of the promoter by C/EBP-α (Miller et al., 1996). Mason et al. (1998) have also shown that mutations in the C/EBP-α and TATA motifs as well as in a consensus Sp1 site of leptin reduced promoter activity by 10, 10 and 2.5-fold, respectively, and abolished binding of these factors. In addition, a common promoter variant of the human leptin gene has been shown to be significantly associated with serum leptin concentration in obese girls (Le Stunff et al., 2000).

The present study was conducted to independently test the association of different gene-specific polymorphisms with traits that are obviously genetically and phenotypically correlated. The appropriateness of correction for multiple testing in this case is unclear and currently controversial (Perneger, 1998). A conservative criterion to control for multiple testing has therefore not been applied to the results presented here. However, if a highly conservative method of correction, such as the Bonferroni adjustment, is applied to the number of independent tests carried out, certain of the significant associations detected may no longer be significant while others would remain unchanged. Some of the associations observed were close to $\alpha = 0.05$, and may turn out to be just Type I errors in nature.

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Table 7.1. Name, ID, type of mutation, location, position and Pubmed accession number of polymorphisms in the leptin gene considered in the present study

Polymorphism	SNP ID	Mutation	Location	Accession no.	Position
AB070368-528	UASMS2	C/T	Promoter	AB070368	528
AB070368-1759	UASMS3	G/C	Promoter	AB070368	1759
AY138588-252	E2JW	T/A	Exon 2	AY138588	252
AY138588-305	R4C	C/T	Exon 2	AY138588	305
BTA236854-600	E2SL	A/G	Exon 2	AJ236854	600
BTA512639-321	A59V	C/T	Exon 3	AJ512639	321
BTA132764-396	E3SL	C/T	Exon 3	AJ132764	396

[a] PIC = polymorphism information content

[b] E2JW and E2SL have only two genotype classes.

Table 7.2. Number of individuals, PIC, Heterozygosity, and allele frequency of each SNP

Locus Name	Individuals	PIC[a]	Heterozygosity	Alleles	Frequency (± SE)
UASMS2	464	0.25	0.32	C	0.82 ± 0.01
				T	0.18 ± 0.01
UASMS3	464	0.37	0.48	C	0.40 ± 0.02
				G	0.60 ± 0.02
E2JW[b]	464	0.31	0.51	A	0.26 ± 0.01
				T	0.74 ± 0.01
R4C	464	0.37	0.51	C	0.42 ± 0.02
				T	0.58 ± 0.02
E2SL[b]	464	0.07	0.08	A	0.96 ± 0.01
				G	0.04 ± 0.01
A59V	464	0.31	0.38	C	0.26 ± 0.01
				T	0.75 ± 0.01
E3SL	464	0.30	0.40	C	0.24 ± 0.01
				T	0.77 ± 0.01

Table 7.3. Chi-square test statistics for Hardy-Weinberg equilibrium (on diagonal) and linkage disequilibrium (LD, off-diagonal) among different leptin SNP.

SNP	UASMS2	UASMS3	E2JW	R4C	E2SL	A59V	E3SL
UASMS2	1.228	54.54***	0.001	24.29**	5.02*	29.83**	188.15**
UASMS3		0.002	15.64**	366.2***	15.383**	111.5***	52.08***
E2JW			54.58***	18.91**	1.49	7.76*	0.91
R4C				0.75	17.35**	117.5***	69.76***
E2SL					0.76	1.44	2.84
A59V						0.05	51.54**
E3SL							5.00*

* significant LD ($P < 0.05$); ** significant LD ($P < 0.01$); *** significant LD ($P < 0.001$)

Table 7.4. Number of animals with records, mean, and SD for traits considered in the commercial population (n = 144).

Trait	Mean	SD
Residual feed intake, kg/d	0.00	0.66
Feed conversion ratio, kg DM/kg gain	5.67	0.63
Daily DMI, kg/d	8.17	1.05
Metabolic BW, kg$^{0.75}$	87.76	7.74
Average daily gain, kg/d	1.57	0.25
Gain in backfat, mm/d	0.06	0.04
Gain in ultrasound LM area, cm^2/d	0.04	0.03
Ultrasound backfat, mm	8.95	3.01
Ultrasound LM area, cm^2	85.52	8.84
Ultrasound marbling score	5.19	0.70
Carcass weight, kg	293.52	44.47
Cecass LM area, cm^2	76.64	9.88
Carcass grade fat, mm	9.81	3.87
Carcass marbling score	445.99	59.29
Carcass yield grade	1.64	0.65
Carcass lean meat yield, %	57.81	3.06

Table 7.5. Effect of R4C SNP in leptin exon 2 on measures of feed efficiency, performance, ultrasound and carcass merit of steers from a commercial population of beef cattle (n = 144).

Traits	R4C SNP genotype				
	CC	CT	TT	SED[d]	*P* value
Number of steers	32	74	38		
Residual feed intake, kg/d	-0.18	0.05	0.19	0.05	0.23
Feed:gain ratio, kg DM/kg gain	5.66	5.66	5.77	0.05	0.70
DM intake, kg/d	8.16	8.23	8.25	0.09	0.88
Metabolic BW, kg$^{0.75}$	87.54	88.74	88.39	0.64	0.43
Average daily gain, kg/d	1.57	1.57	1.54	0.02	0.88
Gain in backfat, mm/d	0.06^{c}	0.07^{b}	0.09^{a}	0.003	0.02
Ultrasound backfat, mm	8.66	8.79	10.11	0.25	0.07
Ultrasound LM area, cm^2	85.72	84.85	84.84	0.71	0.90
Ultrasound marbling score	5.27	5.13	5.31	0.06	0.54
Carcass weight, kg	290.66	292.33	295.14	3.71	0.61
LM area, cm^2	76.09	76.27	74.52	0.82	0.74
Grade fat, mm	8.65^{c}	10.10^{b}	11.47^{a}	0.32	0.005
Marbling score[e]	442.20	4452	442.18	4.94	0.74
Lean meat yield, %	58.58^{a}	57.64^{a}	56.25^{b}	0.26	0.007
Yield grade	1.52^{b}	1.64^{b}	2.00^{a}	0.055	0.01

[a, b, c] Least squares means in rows followed by different superscripts are different

[d] Standard error of the difference between least squares means.

[e] Marbling score is a measure of intramuscular fat: trace marbling or less = 100 to 399 (Canada A quality grade); slight marbling = 400 to 499 (Canada AA quality grade); small to moderate marbling = 500 to 799 (Canada AAA quality grade); slightly abundant or more marbling = 800 to 1100 (Canada Prime).

Table 7.6. Effect of R4C SNP in leptin exon 2 with measures of serum leptin concentration, performance, feed efficiency, ultrasound, and carcass merit in composite cattle (n = 464).

	R4C SNP genotype			
Trait	CC	CT	TT	*P* value[a]
Number of animals	152	235	77	
Serum leptin, ng/mL	13.67 ± 0.47	13.67 ± 0.38	13.30 ± 0.63	0.89
Phenotypic RFI, kg/d	-0.05 ± 0.10	-0.03 ± 0.06	-0.03 ± 0.08	0.99
Genetic RFI, kg/d	-0.18 ± 0.10	-0.19 ± 0.06	-0.17 ± 0.07	0.97
Feed conversion, kg DM/kg gain	7.22 ± 0.16	7.21 ± 0.11	7.34 ± 0.13	0.50
DM intake, kg/d	10.60 ± 0.18	10.52 ± 0.13	10.50 ± 0.15	0.86
Metabolic BW, kg$^{0.75}$	91.48 ± 1.00	90.99 ± 0.80	91.48 ± 0.87	0.67
Average daily gain, kg/d	1.51 ± 0.04	1.49 ± 0.03	1.46 ± 0.03	0.39
Ultrasound backfat, mm	8.59 ± 0.25^{c}	9.02 ± 0.20^{bc}	9.59 ± 0.34^{b}	0.049
Ultrasound LM area, cm^2	84.01 ± 0.66	83.04 ± 0.51	82.02 ± 0.87	0.17
Ultrasound marbling score	5.14 ± 0.07	5.13 ± 0.06	5.12 ± 0.09	0.97
Number of animals	122	196	63	
Carcass weight, kg	315.9 ± 3.7	310.8 ± 3.2	313.3 ± 4.4	0.26
Carcass grade fat, mm	10.25 ± 0.35^{c}	10.31 ± 0.28^{c}	11.54 ± 0.48^{b}	0.06
Average backfat, mm	11.72 ± 0.36^{c}	11.80 ± 0.28^{c}	12.90 ± 0.48^{b}	0.09
LM area, cm^2	85.43 ± 0.85^{b}	83.51^{bc} ± 0.71	82.16 ± 1.07^{c}	0.026
Carcass marbling score	2.50 ± 0.05	2.45 ± 0.05	2.47 ± 0.07	0.63
Lean meat yield	58.46 ± 0.37^{b}	58.15 ± 0.30^{bc}	57.16 ± 0.48^{c}	0.07
Flight speed, m/s	2.71 ± 0.08^{b}	2.43 ± 0.07^{c}	2.35 ± 0.11^{c}	0.007

[a] *P* value from overall F test

[b, c, d] Means in rows followed by different superscripts are different ($P < 0.05$)

Table 7.7. Association of UASMS2 SNP in leptin promoter (LS means ± SE) with measures of serum leptin concentration, performance, feed efficiency, feeding behaviour, and carcass merit in composite cattle (n = 150).

Trait	UASMS2 SNP genotype CC	CT	TT	*P* value[a]
Number of animals	99	45	6	
Serum leptin level, ng/mL	11.97 ± 0.82^{d}	14.10 ± 0.96^{c}	23.22 ± 2.44^{b}	< 0.001
Dry matter intake, kg/d	9.62 ± 0.20^{c}	10.53 ± 0.21^{b}	10.19 ± 0.53^{b}	0.001
Phenotypic RFI, kg/d	-0.43 ± 0.19^{b}	-0.08 ± 0.21^{c}	-0.63 ± 0.38^{b}	0.06
Feed conversion, kg DM/kg gain	7.36 ± 0.15	7.25 ± 0.16	7.28 ± 0.41	0.81
Metabolic size, kg$^{.75}$	85.13 ± 1.60^{c}	87.51 ± 1.77^{c}	91.40 ± 2.26^{b}	0.05
Average daily gain, kg/d	1.33 ± 0.03^{c}	1.47 ± 0.04^{b}	1.42 ± 0.10^{b}	0.002
Final BW, kg	483.12 ± 11.6^{d}	499.69 ± 12.7^{c}	527.71 ± 23.51^{b}	0.10
Feeding duration, min/d	51.54 ± 2.34^{c}	56.95 ± 2.58^{b}	52.38 ± 4.77^{c}	0.02
Feeding frequency, events/d	36.48 ± 1.56	34.78 ± 1.71	32.37 ± 3.16	0.22
Ultrasound backfat, mm	5.59 ± 0.48^{d}	6.30 ± 0.51^{c}	9.19 ± 1.01^{b}	0.001
Ultrasound marbling score	4.63 ± 0.12^{d}	4.82 ± 0.13^{c}	5.30 ± 0.25^{b}	0.01
Ultrasound LM area, cm^2	73.05 ± 1.45	72.17 ± 1.60	70.03 ± 2.94	0.40

[a] *P* value from overall F test

[b, c, d] Means in rows followed by different superscripts are different ($P < 0.05$)

Table 7.8. Association of UASMS2 SNP in the leptin promoter (LS means ± SE) with measures of serum leptin concentration, performance, feed efficiency, ultrasound, and carcass merit in composite crossbred cattle (n = 464)

Trait	UASMS2 SNP genotype			*P* value[a]
	CC	CT	TT	
Number of animals	306	146	12	
Serum leptin level, ng/mL	13.04 ± 0.38^{c}	13.94 ± 0.54^{c}	19.20 ± 1.53^{b}	<0.001
Phenotypic RFI, kg/d	-0.07 ± 0.07^{c}	0.16 ± 0.09^{b}	0.13 ± 0.26^{b}	0.09
Genetic RFI, kg/d	-0.21 ± 0.07^{c}	0.01 ± 0.09^{b}	0.04 ± 0.26^{b}	0.10
Feed conversion, kg DM/kg gain	7.21 ± 0.11	7.37 ± 0.14	7.22 ± 0.33	0.47
Dry matter intake, kg/d	10.33 ± 0.13^{c}	10.71 ± 0.17^{bc}	11.09 ± 0.42^{b}	0.036
Average daily gain, kg/d	1.47 ± 0.03	1.45 ± 0.03	1.53 ± 0.08	0.52
Metabolic BW, $kg^{0.75}$	90.65 ± 0.68	90.96 ± 0.83	92.58 ± 1.84	0.56
Backfat gain, mm/d (x 10^{-2})	3.40 ± 0.10^{c}	3.33 ± 0.10^{c}	4.60 ± 0.40^{b}	0.017
Marbling gain, units/d (x 10^{-2})	0.70 ± 0.02^{c}	0.70 ± 0.04^{c}	1.00 ± 0.10^{b}	0.05
Ultrasound backfat, mm	8.93 ± 0.20^{d}	9.09 ± 0.28^{c}	11.38 ± 0.83^{b}	0.017
Ultrasound marbling score	5.07 ± 0.06^{c}	5.22 ± 0.08^{b}	5.58 ± 0.20^{b}	0.023
Ultrasound LM area, cm^2	83.61 ± 0.56	83.83 ± 0.80	80.73 ± 2.29	0.42
Number of animals	255	118	8	
Carcass grade fat, mm	10.32 ± 0.30^{c}	10.58 ± 0.44^{c}	13.37 ± 1.36^{b}	0.09
Average carcass backfat, mm	11.82 ± 0.29^{c}	12.13 ± 0.43^{c}	14.82 ± 1.36^{b}	0.09
LM area	84.18 ± 0.72	83.71 ± 0.98	82.86 ± 2.75	0.83
Carcass marbling score	2.47 ± 0.04	2.54 ± 0.06	2.65 ± 0.17	0.35
Lean meat yield	58.17 ± 0.17	57.98 ± 0.44	55.88 ± 1.26	0.17

[a] *P* value from overall F test

[b, c, d] Means in rows followed by different superscripts are different ($P < 0.05$).

Table 7.9. Association of A59V SNP in leptin exon 3 (LS means ± SE) with measures of serum leptin concentration, performance, feed efficiency, feeding behaviour, and carcass merit in composite crossbred cattle.

Trait	A59V SNP genotypes			*P* value[a]
	CC	CT	TT	
Number of animals	31	174	259	
Serum leptin level, ng/mL	10.80 ± 0.98^{d}	13.40 ± 0.40^{c}	14.43 ± 0.37^{b}	0.0029
Phenotypic RFI, kg/d	0.03 ± 0.16	-0.02 ± 0.07	-0.06 ± 0.06	0.79
Genetic RFI, kg/d	-0.04 ± 0.06	-0.16 ± 0.06	-0.22 ± 0.06	0.59
Dry matter intake, kg/d	10.33 ± 0.25	10.53 ± 0.14	10.55 ± 0.14	0.70
Average daily gain, kg/d	1.36 ± 0.05^{c}	1.48 ± 0.03^{b}	1.50 ± 0.03^{b}	0.039
Metabolic BW, kg$^{0.75}$	91.28 ± 1.31	91.35 ± 0.84	91.11 ± 0.83	0.93
Feed conversion, kg DM/kg gain	7.96 ± 0.23^{b}	7.26 ± 0.12^{c}	7.20 ± 0.12^{c}	0.005
Partial efficiency of growth	0.27 ± 0.009^{d}	0.29 ± 0.004^{c}	0.30 ± 0.003^{b}	0.06
Relative growth rate (x 10^{-2})	14.8 ± 0.59^{c}	16.23 ± 0.25^{b}	16.44 ± 0.24^{b}	0.013
Kleiber ratio, (x 10^{-2})	1.48 ± 0.05^{c}	1.62 ± 0.03^{b}	1.72 ± 0.02^{b}	0.013
Ultrasound backfat, mm	7.93 ± 0.55^{d}	8.74 ± 0.23^{c}	9.46 ± 0.21^{b}	0.014
Ultrasound LM area, cm^{2}	82.20 ± 1.42^{c}	84.55 ± 0.61^{b}	83.24 ± 0.57^{bc}	0.011
Number of Animals	*26*	*143*	*212*	
Carcass grade fat, mm	9.52 ± 0.79	10.15 ± 0.36	10.94 ± 0.33	0.10
Average carcass backfat, mm	10.63 ± 0.78^{d}	11.64 ± 0.34^{c}	12.55 ± 0.30^{b}	0.039
Carcass LM area, cm^{2}	84.40 ± 1.64^{bc}	85.56 ± 0.81^{b}	83.83 ± 0.75^{c}	0.015
Carcass marbling score	2.45 ± 0.10	2.44 ± 0.05	2.51 ± 0.05	0.47
Lean meat yield	59.13 ± 0.74^{b}	58.56 ± 0.34^{bc}	57.47 ± 0.31^{c}	0.024
Carcass yield grade	1.67 ± 0.14	1.59 ± 0.06	1.76 ± 0.06	0.10

[a] *P* value from overall F test

[b, c, d] Means in rows followed by different superscripts are different ($P < 0.05$).

Table 7.10. Association of UASMS2 and A59V genotype combinations with serum leptin concentration, performance, feed efficiency, ultrasound, and carcass merit in crossbred composite cattle.

Trait[a]	UASMS2 and A59V haplotype						SEM	Effect[b], %	*P* value[b]
	CCCC	CCCT	CCTT	CTCT	CTTT	TTTT			
Animals	31	127	148	47	99	12	–	–	–
SLPT	10.37^{e}	12.91^{de}	14.10^{d}	14.01^{d}	14.11^{d}	19.39^{c}	0.80	8.97	<0.001
RFIp	0.02	-0.04	-0.16	0.06	0.07	0.11	0.13	0.13	0.44
RFIg	-0.06	-0.19	-0.31	-0.09	-0.07	-0.06	0.13	0.03	0.69
DMI	10.32	10.47	10.48	10.70	10.63	10.91	0.22	0.16	0.29
ADG	1.36^{d}	1.47^{c}	1.51^{c}	1.46^{c}	1.47^{c}	1.51^{c}	0.04	0.87	0.042
MWT	91.21	91.02	91.04	92.38	91.22	92.12	1.20	0.01	0.70
FCR	7.89^{c}	7.24^{d}	7.08^{d}	7.49^{dc}	7.42^{dc}	7.22^{d}	0.22	0.50	0.019
RGR (x 10^{-2})	14.85^{d}	16.18^{cd}	16.54^{c}	15.66^{cd}	15.85^{cd}	16.46^{c}	0.40	1.24	0.0028
KRAT (10^{-2})	1.48^{d}	1.65^{c}	1.64^{c}	1.57^{cd}	1.58^{cd}	1.65^{c}	0.09	1.19	0.0053
UBF	7.82^{e}	8.55^{de}	9.42^{d}	9.16^{d}	9.07^{d}	11.38^{c}	0.43	6.68	0.005
UMAR	5.15^{e}	5.02^{e}	5.11^{e}	5.26^{de}	5.20^{e}	5.55^{c}	0.11	6.26	<0.001
ULMA	82.32^{d}	84.07^{cd}	82.19^{d}	85.71^{c}	82.96^{d}	81.64^{d}	1.13	0.91	0.01
Animals	*26*	*109*	*120*	*34*	*84*	*8*	–	–	–
CGF, mm	9.66^{e}	9.89^{de}	10.89^{d}	10.84^{d}	10.55^{d}	13.51^{c}	0.69	5.53	<0.001
CBF, mm	10.75^{e}	11.40^{de}	12.40^{d}	12.27^{d}	12.18^{d}	15.12^{c}	0.70	5.84	<0.001
CMAR	2.42^{e}	2.41^{e}	2.48^{de}	2.56^{cd}	2.51^{cd}	2.72^{c}	0.09	4.16	<0.001
CREA	84.33^{cd}	85.16^{cd}	82.60^{d}	86.09^{c}	82.86^{d}	83.70^{d}	1.42	4.85	<0.001
CYG	1.67	1.57	1.74	1.72	1.65	2.04	0.12	4.20	<0.001
LMY	59.07^{c}	58.70^{cd}	57.53^{d}	58.08^{cd}	57.80^{d}	55.59^{e}	0.65	8.92	<0.001

[a] SLPT = serum leptin concentration (ng/mL); RFIp = phenotypic RFI (kg/d); RFIg = genetic RFI (kg/d); DMI = daily dry matter intake ((kg/d); MWT = metabolic BW ($kg^{0.75}$); ADG = average daily gain (kg/d); FCR = feed conversion ratio (kg DM/kg gain); RGR = relative growth rate; KRAT = Kleiber ratio; UBF = ultrasound backfat (mm); UMAR = ultrasound marbling score; ULMA = ultrasound LM area (cm^2); CGF = carcass grade fat (mm); CBF = carcass backfat (mm); CMAR = carcass marbling; CLMA = carcass LM area (cm^2); CYG = carcass yield grade; LMY = lean meat yield (%).

[b] *P* values and haplotype effects are from haplotype regression using dummy variables. Haplotype effects are expressed as % of total phenotypic variation in the trait. shown.

[c, d, e] Means in rows followed by different superscripts are different ($P < 0.05$).

CHAPTER 8

Genome-wide search for QTL affecting feed intake, feed efficiency, feeding behaviour, and temperament in composite cattle.

8.1 Introduction

Feed intake and feed efficiency of beef cattle have been identified as economically relevant traits (**ERT**), which have a large impact on the production cost, profitability, global competitiveness, and environmental sustainability of the beef industry. Recently, there have been considerable research efforts in a number of countries aimed at genetically improving the overall biological and economic efficiency of the beef production system, including the consideration of those characteristics that determine consumer acceptability of the final product. The application of genomic information obtained through quantitative trait loci (**QTL**) mapping studies in genetic marker-assisted selection will complement and greatly accelerate the rate of genetic progress towards improvement in beef cattle productivity, efficiency and environmental sustainability.

Feed intake and feed efficiency traits are however, very difficult and expensive to measure. Therefore, despite recent world-wide efforts to genetically improve beef cattle feed efficiency (Arthur et al., 2001; Herd et al., 2003; Crews et al., 2005) there have been very few attempts at identifying QTL for these traits (Pitchford et al., 2002; Nkrumah et al., 2005). Previous QTL mapping projects in beef cattle have mainly concentrated on production (Elo et al., 1999; Casas et al., 2000; Grosz and MacNeil, 2001; Kneeland et al., 2004) or carcass and meat quality traits (Casas et al., 2000; Kim et al., 2003; Moore et al., 2003; Li et al., 2004).

In addition to feed intake and feed efficiency, information on animal behaviour and temperament traits may be incorporated into genetic evaluations either because of correlated responses as a result of selection for an ERT or because the behaviour trait may have a direct economic value. Previous QTL mapping studies on behaviour traits in cattle have been reported by Schmutz et al. (2001) and Hiendleder et al. (2003). Genomic information on behaviour traits from QTL mapping studies may be incorporated into breeding programs to accelerate the rate of genetic improvement in beef cattle productivity, efficiency, animal handling and herd management safety. In the present study, we employed a multiple marker regression interval mapping approach to comprehensively scan the bovine autosomal genome to identify chromosomal regions harbouring QTL for feed intake, feed efficiency, feeding behaviour, and temperament.

8.2 Materials and Methods

8.2.1 *Animals and Phenotypic Data*

Growth, feed intake, feeding behaviour, and temperament data collected between 2002 and 2005 on 464 composite crossbred beef steers were used in this study. The steers were sired by Angus, Charolais or University of Alberta Hybrid bulls and belonged to 28 paternal half-sib families. The resource population of dams used in the study was produced from crosses among three composite cattle lines, namely Beef Synthetic 1, Beef Synthetic 2, and Dairy X Beef Synthetic (Goonewardene et al., 2003). Cows and heifers for the study were bred in multiple sire breeding groups on pasture and the sire of each calf was later determined in a parentage test using a panel of bovine microsatellite markers.

Details of the procedures for the feedlot tests were given by Nkrumah et al. (2004) and are also shown in Chapter 3. Briefly, the animals were managed and tested under feedlot conditions using the GrowSafe automated feeding system (GrowSafe Systems Ltd., Airdrie, Alberta, Canada) at the University of Alberta's Kinsella Research Station. The animals weighed 353.0 (SD = 61.3) kg and were 252 (SD = 42) days of age at the beginning of testing. Two tests using approximately 80 animals per test were conducted each year. The animals used in the study were cared for according to the guidelines of the Canadian Council on Animal Care (CCAC, 1993).

Feed intake and efficiency traits analyzed include feedlot average daily **DMI**, feed conversion ratio (**FCR**), residual feed intake (**RFI**), and partial efficiency of growth (**PEG**). The feedlot behaviour traits studied were daily feeding duration (**FD**), daily feeding "head down" time (**HD**), daily feeding frequency (**FF**) and flight speed (**FS**) as a measure of temperament. Procedures for obtaining the measures of feedlot feed efficiency have been described previously (Nkrumah et al., 2004). Linear regression in SAS (SAS Institute, Inc., Cary, NC, version 9.1.3) of weekly or fortnightly BW against time (d) was used to derive ADG and mid-test metabolic size (MWT, $BW^{0.75}$) for each steer. The total feed intake of each animal over a 70-d test period was used to compute the daily DMI. Feed conversion ratio was computed as the ratio of DMI to ADG on test. Residual feed intake is an alternative measure of feed efficiency, and was calculated from phenotypic regression (phenotypic RFI, Arthur et al., 2001) or genetic regression (genetic RFI, Crews et al., 2005; Hoque et al., 2005) of ADG and MWT on DMI. The partial efficiency of growth (PEG; i.e., energetic efficiency for ADG above maintenance) of each animal was computed as the ratio of ADG to the difference between average daily DMI and expected DMI for maintenance (**DMIm**) (Arthur et al., 2001), where DMIm was computed according to NRC (1996).

Procedures for determining the feeding behaviour traits from the GrowSafe System have previously been described (Basarab et al., 2003). Daily feeding duration was computed as the sum of the difference between feeding event end-times and start-times per day for each animal. It was equal to the total number of minutes each day spent in feeding and feeding-related activities (prehension, chewing, backing away from the bunk and chewing, socializing, scratching, or licking) at the feedbunk. Head down time refers to the sum of the number of times the animal's electronic identification (transponder) was detected by the GrowSafe System during a feeding event, multiplied by the system's scanning time, where scanning time is system dependent and ranges from 1.0 to 6.3 s. Daily feeding frequency in this study was defined as the number of independent feeding events for a particular animal in a day. A feeding event starts when an animal's transponder is first detected and ends when the time between the last two transponder readings on that animal was greater than 300 s or when a different transponder number is encountered. Flight speed was calculated from the time in seconds taken to traverse a fixed distance of 2.44 m after exiting a squeeze chute. Infrared sensors were used to trigger the start and stop of the timing system (Burrow et al., 1988).

8.2.2 *DNA Isolation and Genotyping*

A 10-mL blood sample was collected by jugular venipuncture from each animal at the start of the feed efficiency tests from which genomic DNA was extracted using a standard saturated salt phenol/chloroform procedure (Miller et al., 1988). A whole-genome scan covering all 29 *Bos taurus* (**BTA**) autosomes was performed with 455 genetic markers. The marker panel used to genotype the 464 steers and their sires consisted of 100 microsatellite and 355 single nucleotide polymorphisms (**SNP**). The microsatellite markers were selected from the USDA MARC website (http://www.marc.usda.gov/cattle), whereas the SNP markers were chosen from an on-going whole-genome bovine SNP RH mapping project (McKay et al., 2005). The markers were chosen to be roughly evenly distributed across all 29 autosomes. In addition to chromosomal position, criteria for selection of microsatellite markers were polymorphism information content, sire heterozygosity, number of alleles, and ease of scoring. The criteria for choosing the SNP markers were location and sire heterozygosity.

The locations of the microsatellite markers were based on the most up-to-date linkage map locations of these markers as presented by Snelling et al. (2005). The approximate linkage map locations of the SNP markers were inferred from the linkage map locations of the nearest microsatellite markers (with known RH map and linkage map locations) from recently created bovine RH maps for the respective chromosomes. The 455 markers spanned approximately 2813.5 cM of the bovine autosomal genome, with an average marker spacing of 6.18 cM and a range of 3.13 cM for BTA 28 to 9.68 cM for BTA 1. The number of markers per chromosome averaged 16 and ranged from 7 on BTA 26 to 33 on BTA 5. Microsatellite marker genotypes were determined by automated fragment analysis using the ABI PRISM 377 and ABI 3730 DNA sequencers (Applied Biosystems, Foster City, CA). A second examiner evaluated all microsatellite marker genotypes.

Genotyping of all SNP used in the study was carried out using the Illumina GoldenGate assay on the BeadStation 500G Genotyping System (Illumina Inc., San Diego, CA). The GoldenGate assay uses an allele-specific extension reaction and universal PCR technology to multiplex and genotype simultaneously up to 1,536 SNP loci from approximately 250 ng genomic DNA (Oliphant et al., 2002; Shen et al., 2005). All marker data were checked for typing errors through an examination of Mendelian segregation. The

PROC ALLELE of SAS/Genetics 9.1.3 (SAS Institute, Inc., Cary, NC) was used to obtain summaries of allele and genotype frequencies, polymorphism information content (**PIC**), heterozygosity, and other measures of marker informativeness for each marker using maximum likelihood estimation.

8.2.3 *Analysis of Phenotypic Data*

Prior to the QTL analyses, the phenotypic data were adjusted for a number of systematic effects by assuming a polygenic inheritance model containing nongenetic effects of year (three levels), contemporary group nested within year (two levels per year), age of dam, and age of steer on test. All 464 animals were used in this step. The statistical model to describe each given phenotypic observation **Y**, for each trait was:

$$Y = X\beta + Zu + e$$

where β is a vector of fixed effects and the regression coefficients for dam age and age of steer on test, $\boldsymbol{X}$ is a known incidence matrix relating observations to their fixed effect levels and the values for covariables dam age and age of steer on test. Vector $\boldsymbol{u}$ contains polygenic effects for all animals in the dataset pedigree, which are linked to observations $\boldsymbol{y}$ by the incidence matrix $\boldsymbol{Z}$. Vector **e** contains random residual errors. Subsequently, the trait score for the interval mapping analyses, $\boldsymbol{y}^*$, contains the phenotypes corrected for the nongenetic effects estimated under the above model as:

$$y* = Y - X\hat{b}$$

The GLM procedure of SAS 9.1.3 (SAS Institute, Inc., Cary, NC) was used to carry out these estimations for each trait separately.

8.2.4 *Genome-wide search for QTL*

Twelve out of the 28 families had eight offspring or less and were excluded from the QTL analysis, in order to increase the power of detection. The number of steers in each of the remaining 16 families ranged from 10 to 56 and averaged 37. Quantitative trait loci were detected using a multiple marker regression approach for half-sib families as described by Knott et al. (1996), which was applied to QTL mapping studies in cattle by Spelman et al. (1996) and Vilkki et al. (1997), and implemented in the web-based software

QTL Express (Seaton et al., 2001, http://qtl.cap.ed.ac.uk/arabica[illegible]ervlets/hkloader?HS). The method contains the following steps: The paternal alleles inherited by each progeny are identified for all markers for which the sire is heterozygous (i.e. informative). The procedure then determines the most likely phases of the gametes of the sire of each family (the phase that minimizes the number of recombination events in the offspring).

For a given informative marker, the allele inherited by the offspring is unambiguously determined if the son possessed only one of the sire's alleles. If the offspring is heterozygous for the same alleles as the sire, then the most likely paternal haplotype was estimated from the nearest informative flanking marker by choosing the linkage phase that minimizes the number of offspring that are recombinant for the particular interval. One of the haplotypes of the sire was arbitrarily chosen as the first haplotype. Then for each offspring, the conditional Identical-By-Descent **(IBD)** probability that it inherited the first allele (haplotype) of a putative QTL are derived at 1 cM intervals using the smallest informative marker interval available for each sire on each chromosome (George et al., 2000). Subsequently, an F-statistic for H_0 (no QTL) vs. H_A (one QTL) for a QTL with a gene substitution effect was fitted at 1 cM intervals along each chromosome using the model:

$$y^*_j = a_i + b_i X_{ij} + e_{ij}$$

where y^*_j is the adjusted trait score of individual j, originating from sire i; a_i is the average effect for half-sib family i; b_i is the regression coefficient within half-sib family i (*i.e.*, substitution effect for a putative QTL, Falconer and Mackay, 1996); X_{ij} is the IBD conditional probability for individual j of inheriting the first parental haplotype of a putative QTL, and e_{ij} is the residual effect. The regression is nested within families since the assignment of the first gamete is random and not all sires are heterozygous for the QTL. Both within- and across-family analyses were conducted for each trait along each chromosome. Significance thresholds from an empirical distribution of the test statistic under the null hypothesis of no QTL associated with the chromosome under study were determined by permutations as described by Churchill and Doerge (1994). For the present study 2000 permutations (across-family) and 2500 permutations (within-family) were studied to determine the chromosome-wise 10, 5, and 1% significance thresholds.

8.3 Results and discussion

Table 8.1 gives an overview of the phenotypic characteristics of the traits considered in the present study. Heritability estimates ranged from 0.21 to 0.59. The traits were evaluated using genotype information from 455 markers on 370 steers across and within 16 families to identify QTL affecting feed intake, feed efficiency, feeding behaviour, and temperament. The across-family test statistic gave an indication of whether a QTL was segregating in the experimental population for the tested linkage group. Since the gene substitution effects are nested within sire families, no assumptions are made regarding the phase of QTL effects in the across-family analyses. The within-family analyses correspond to a contrast of the two sire haplotypes, and give an indication of the families that are heterozygous for the QTL. The linkage phase between a marker and a QTL can differ among families. Additionally, in a half-sib model, the most likely position of a putative QTL across families is not necessarily the most likely position of the QTLs within families, mainly due to among-family variations in marker informativeness (de Koning et al., 1999). Several QTLs were detected in both the within- and across-family analysis that exceeded the chromosome-wise *P* values of < 0.05 or < 0.01. The across-family analysis revealed five QTLs on two chromosomes, 15 QTLs on 10 chromosomes, and 21 QTLs on 14 chromosomes that exceeded the chromosome-wise probability thresholds of 0.01, 0.05, and 0.10, respectively (Table 8.2). On the other hand, there were 51 and 182 QTLs effects that exceeded the chromosome-wise $P = 0.01$ and 0.05 thresholds, respectively in the within-family analysis (Tables 8.3 – 8.11). Figures 8.1 – 8.4 show the QTL profiles of feed intake and feed efficiency traits whiles Figures 8.5 – 8.10 show profiles of QTLs for feeding behaviour and temperament traits observed.

Four significant effects on four different chromosomes (chromosome-wise $P < 0.05$) were detected for DMI in the across-family analysis (Table 8.2). For DMI, a total of 29 tests of hypothesis were conducted with one significant test expected by chance at $P = 0.05$, and four were observed. A total of 32 significant QTLs effects on 20 different chromosomes were detected for DMI that exceeded the chromosome-wise $P = 0.05$ threshold (Table 8.3) in the within-family analysis. Of these, eight QTLs on eight different chromosomes were significant at the chromosome-wise $P < 0.01$ threshold. In the within-family analysis, a total of 464 tests were carried out with 23 and four expected by chance at the chromosome-wise P = 0.05 and 0.01 levels, and 32 and eight were detected, respectively. The most significant QTLs for DMI were detected on BTA 3, 8, 10, 12, 15, 20, 28, and 29, with the highest being the BTA 20 QTL. The QTL allele substitution effects for DMI averaged 1.78 and ranged from 0.92 to 3.80 genetic standard deviations. One QTL on BTA had a rather large effect of 5.79 genetic standard deviations.

Two significant (chromosome-wise $P < 0.05$) and two suggestive (Chromosome-wise $P < 0.10$) QTLs were detected for FCR in the across family analysis. One QTL was expected by chance at $P = 0.05$ based on the number of tests carried out, and two were detected. In the within-family analysis (Table 8.4), a total of 28 significant ($P < 0.05$) QTL effects were detected on 17 different chromosomes, of which five exceeded the chromosome-wise $P = 0.01$ threshold. A total of 23 false positives are expected by chance at $P = 0.05$, and 28 were observed. The QTL effects averaged 2.25 and ranged from 0.95 to 6.79 genetic standard deviations. The strongest FCR QTL was detected on BTA 23 (F statistic = 14.15) at 59 cM. The same region of BTA 23 has previously been shown to habour QTL for growth rate (Kneeland et al., 2004). In the present dataset, a QTL for ADG was detected at the beginning of the same linkage group (data not shown). Indeed, several of the FCR QTLs in the present study were located in regions that were also significant or suggestive for ADG QTL. Approximately the same regions (± 5 cM) of BTA 1, 2, 6, 10, 14, 15, 23, and 27, which were significant for FCR QTLs were also significant for QTLs influencing ADG. This is not surprising as several studies have shown that there is a strong genetic correlation between ADG and FCR in beef cattle (Herd et al., 2003; Crews et al., 2005). In the present population, the genetic correlation of FCR with ADG was -0.59.

Two measures of residual feed intake, (genetic and phenotypic RFI) were evaluated for QTL effects. The genetic and phenotypic correlations between these two indices of RFI are both greater than 0.90 in the present study as well as in the studies by Hoque and Oikawa (2004) and Hoque et al. (2005). Indeed, all the QTLs detected for phenotypic RFI were also significant for genetic RFI, albeit slight variations in number of heterozygous families and QTL locations, with the exception of two QTLs on BTA12 and 23 for phenotypic RFI and BTA 18 and 22 for genetic RFI, which were not common between the two indices (Tables 8.5 and 6). The across-family analysis revealed one QTL on BTA 20 that was significant for both genetic and phenotypic RFI at the chromosome-wise $P < 0.01$ threshold, with less than one QTL expected by chance out of 29 tests. Two QTLs for genetic RFI and one QTL for phenotypic RFI were significant at the chromosome-wise $P < 0.05$ threshold in the across-family analysis. Additionally, five and four suggestive (chromosome-wise $P < 0.10$) QTLs were detected for phenotypic and genetic RFI, respectively, in the across-family analysis. Combining those QTL that are not common to the two RFI indices, there were a total of 31 significant (chromosome-wise $P < 0.05$) QTL effects on 16 different chromosomes in the within-family analysis, of which eight exceeded the chromosome-wise $P = 0.01$ threshold. The most significant QTL for both measures of RFI occurred on BTA 20. The RFI QTL detected on BTA 2, 10, 14, 15, and 23 occurred in the same regions where QTL were detected for ADG. The allele substitution effect for the remaining phenotypic and genetic RFI QTL, respectively, averaged 4.26 and 2.63 (range 1.31 – 9.15 and 0.78 – 6.36) genetic standard deviations.

Partial efficiency of growth is the energetic efficiency for average daily gain. The trait has been shown to be strongly correlated phenotypically (r = -0.65) and genetically (r = 0.94) with RFI. The genetic and phenotypic correlations of PEG with phenotypic RFI were -0.83 and -0.87, respectively. With such strong correlations, it is expected that very similar QTL effects will be observed for PEG as with RFI. A QTL significant at chromosome-wise $P < 0.01$ on BTA 20 and another QTL significant at chromosome-wise $P < 0.05$ on BTA 2 were detected in the across-family analysis. The remaining QTL for PEG in the across-family analysis were suggestive (chromosome-wise $P < 0.10$). There were a total of 19 significant QTL effects ($P < 0.05$) on 12 different chromosomes in the within-family analysis for PEG, of which four exceeded the chromosome-wise $P = 0.01$ threshold (Table 8.7). The PEG QTL on BTA 2, 3, 10, 14, 15, 17, 19, 20, 23, and 26 were mapped to the same region as corresponding QTL for RFI. The QTL for PEG on BTA 1 occurred in the same region as the ADG QTL, whilst the QTL on BTA 25 was located in a region harbouring a QTL for daily DMI. The most significant QTL for PEG was located on BTA 2 (F statistic = 65.72) at the same location and in the same family as a QTL for genetic and phenotypic RFI. The QTL allele substitution effect for PEG averaged 2.16 (range 0.77 – 7.33) genetic standard deviations.

Comparison of the feed intake and feed efficiency QTL results of the present study with other published work is difficult because there have been very limited attempts at identifying QTL for feed intake and feed efficiency in cattle in general. Though, Pitchford et al. (2002) reported QTL for feed intake and associated traits, the results of that study have so far not been made public. However, van Kaam et al. (1999) reported significant QTL for feed intake on chicken chromosome 1 (234 cM), chromosome 2 (41 cM), and chromosome 4 (147 cM). Recently, Minvielle et al. (2005), working with Japanese quail, reported significant QTL for feed intake on chromosome 1 and for feed intake and RFI on chromosome 20.

Though a number of studies have been carried out to estimate genetic parameters for feeding behaviour traits in cattle (Robinson and Oddy, 2004) and sheep (Cammack et al., 2005), there has not been any attempts at identifying QTL for feeding behaviour traits in cattle. Heritability estimates for the feeding behaviour traits in the present study and that reported by Robinson and Oddy (2004) were moderate. A total of six significant QTL (chromosome-wise $P < 0.05$) were detected in the across-family analysis for feeding behaviour traits (two for feeding duration, three for feeding head down time, and one for feeding frequency). An additional three QTL for feeding frequency reached the chromosome-wise suggestive threshold ($P < 0.10$) in the across-family analysis. There were 19 significant QTL effects ($P < 0.05$) on 14 different chromosomes in the within-family analysis for daily feeding duration, of which five exceeded the chromosome-wise $P = 0.01$ threshold. There were 27 significant QTL effects ($P < 0.05$) on 16 different chromosomes for daily feeding head down time in the within-family analysis, of which seven exceeded the chromosome-wise $P = 0.01$ threshold. For daily feeding frequency, there were 21 significant QTL effects ($P < 0.05$) on 16 different chromosomes for the within-family analysis, and one exceeded the chromosome-wise $P = 0.01$ threshold. The QTL allele substitution effects averaged 2.36 (range 1.19 – 5.14), 2.46 (range 0.68 – 4.12), and 1.47 (range 0.63 – 4.52) genetic standard deviations for daily feeding duration, feeding head down time, and daily feeding frequency, respectively.

Burrow et al. (1988) developed flight speed (based on the measurement of the time to cover a fixed distance after exiting a confined area) as an objective measure of temperament in non-restrained animals. Direct heritability estimate for flight speed in the present population (0.49) tended to be higher than that (0.35) reported by Burrow and Corbet (2000), but was similar to that reported (0.40 - 0.44) by Burrow (2001). Three significant (chromosome-wise $P < 0.05$) and one suggestive (chromosome-wise $P < 0.10$) QTL for flight speed were detected in the across-family analysis. For the within-family analysis, there were 13 significant QTL effects ($P < 0.05$) on 10 different chromosomes for flight speed, and one exceeded the chromosome-wise $P = 0.01$ threshold. The most significant QTL for flight speed was detected on BTA 4 (F statistic = 16.85). The QTL allele substitution effects for flight speed averaged 1.27 (range 0.30 – 3.86) in the present study. Hiendleder et al. (2003) identified QTL for temperament in dairy cattle on BTA 5, 18, 29, and XY. In addition, Schmutz et al. (2001) detected QTL for temperament and habituation on six different cattle chromosomes. The putative QTL for behaviour detected by Schmutz et al. (2001) on BTA 14 (19 cM) and 15 (12 cM) may be supported by the flight speed QTL detected on these chromosomes in the present study.

In the present study, several QTL have been detected for feed intake, feed efficiency, feeding behaviour, and temperament. It is likely that the different QTL detected for the different traits may the result of the pleiotropic effects of a few causal genes, obviously due to the strong genetic correlations among the traits. A few of the QTL allele substitution effects were rather extreme (> 5.0 genetic standard deviations) and these must be interpreted with caution. The QTL effects for the remaining traits fall in the range of values published in other studies (de Koning et al., 1999; Olsen et al., 2002). Finally, many of the QTL detected in the within-family analysis were not detected in the across-family analysis, and the most likely QTL position within families is quite different across families for some QTL. The most obvious reason for this is possible differences in marker information between families.

According to Heyen et al. (1999) if the heterozygosity of the QTL in the population is low due to selection, many QTL will be detected in certain linkage groups in the within-family but not in the across-family analysis. Typically, not all families are informative (heterozygous) for a QTL in an outbred design. The estimates of QTL effects and test statistics in the across-family analysis are derived from a combined analysis of families that are heterozygous or homozygous for the QTL (Hiendleder et al., 2003). Since the number of heterozygous sires segregating for each particular QTL detected was generally small (maximum of 4 informative sires per QTL detected in the present study) in relation to the total number of families tested, the combined estimate for the test statistic in an across-family analysis is expected to be considerably lower than in the within-family analysis.

8.4 Literature cited

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Table 8.1. Number of records, overall mean, phenotypic and genetic SD, and direct heritability estimates for the traits considered in the present study

Traits	Animals	Mean	Phenotypic SD	Genetic SD	Heritability
Dry matter intake, kg/d	464	10.45	1.61	1.16	0.54 ± 0.15
Feed conversion ratio, kg DM/kg gain	464	7.29	1.26	0.80	0.41 ± 0.15
Phenotypic residual feed intake, kg DM/d	464	0.00	0.88	0.39	0.21 ± 0.12
Genetic residual feed intake, kg DM/d	464	-0.14	1.01	0.67	0.42 ± 0.15
Partial efficiency of growth	464	0.29	0.06	0.045	0.56 ± 0.16
Daily feeding duration, min/d	464	66.09	18.61	9.85	0.28 ± 0.12
Feeding head down, min/d	464	36.57	11.91	6.59	0.33 ± 0.12
Daily feeding frequency, events/d	464	29.62	10.19	6.96	0.38 ± 0.13
Flight speed, m/s	302	2.52	0.73	0.50	0.49 ± 0.18

Table 8.2. Results from the across-family regression interval mapping for feed intake, feed efficiency, and behavior traits in beef cattle.

BTA	Trait	Position, cM	F-value	Likelihood ratio	Threshold F[a] ($P = 0.05$)	Threshold F[a] ($P = 0.01$)
2	FCR, kg DM/kg gain	93	2.58	39.81	2.49	2.91
2	Phenotypic RFI, kg/d	90	2.17	33.76	2.38	2.79
2	Genetic RFI, kg/d	91	2.61	40.14	2.47	2.98
2	Partial efficiency of growth	87 (9)	2.32	35.98	2.32	2.69
3	Daily DMI, kg/d	54	2.23	34.58	2.20	2.50
4	Daily DMI, kg/d	100	1.89	31.44	2.26	2.63
4	Feeding frequency, events/d	131	2.20	36.29	2.46	3.02
4	Flight speed, m/s	60	3.44	52.04	2.90	3.64
5	FCR, kg DM/kg gain	75	1.84	28.77	2.68	3.13
5	Phenotypic RFI, kg/d	75	1.97	30.69	2.57	2.91
5	Genetic RFI, kg/d	75	2.12	32.97	2.64	3.02
5	Feeding frequency, events/d	73	2.33	36.07	2.85	3.43
6	FCR, kg DM/kg gain	73	2.41	37.34	2.51	2.97
12	Feeding head down, min/d	81	2.19	34.09	2.12	2.56
14	Daily DMI, kg/d	44	2.54	39.23	2.29	2.51
14	Feeding duration, min/d	70	2.09	32.12	2.08	2.49
16	Feeding frequency, events/d	87	2.15	33.46	2.52	3.05
17	Daily DMI, kg/d	67	2.30	35.6	2.20	2.62
17	Phenotypic RFI, kg/d	70	2.06	32.12	2.37	2.79
17	Genetic RFI, kg/d	71	2.05	31.92	2.47	2.85
17	Partial efficiency of growth	83	2.21	34.31	2.26	2.69
18	Phenotypic RFI, kg/d	7	2.03	31.64	2.45	2.92
18	Genetic RFI, kg/d	7	2.21	34.31	2.56	2.97
19	Feeding head down, min/d	111	2.08	32.39	2.08	2.54
20	Daily DMI, kg/d	75	1.95	30.4	2.18	2.58
20	ME Intake, kcal/kg0.75	76	2.63	40.54	2.27	2.61
20	FCR, kg DM/kg gain	74	2.50	38.63	2.46	2.94
20	Phenotypic RFI, kg/d	75	3.08	47.02	2.27	2.65
20	Genetic RFI, kg/d	75	3.19	48.47	2.37	2.77
20	Partial efficiency of growth	75	2.93	44.85	2.16	2.50

[a] QTL effects with F-values greater than chromosome-wise threshold ($P = 0.10$, $P = 0.05$, and $P = 0.01$) are shown. [b] From 2000 permutations.

Table 8.2 continued. Results from the across-family regression interval mapping for feed intake, feed efficiency, and behavior traits in beef cattle

BTA	Trait	Position, cM	F-value[a]	Likelihood ratio	Threshold F[b] ($P = 0.05$)	Threshold F[b] ($P = 0.01$)
23	Feeding frequency, events/d	50	2.50	38.59	2.43	2.97
24	Daily DMI, kg/d	6	2.17	33.71	2.23	2.71
24	Phenotypic RFI, kg/d	6	2.27	35.15	2.25	2.77
24	Genetic RFI, kg/d	6	2.24	34.79	2.18	2.77
24	Flight speed, m/s	64	3.09	47.14	3.08	4.14
26	Feeding duration, min/d	16	1.85	29.01	1.82	2.21
26	Feeding head down, min/d	16	1.91	29.84	1.87	2.15
26	Flight speed, m/s	38	2.67	41.13	2.63	2.87
28	Flight speed, m/s	41	3.25	49.36	3.45	4.54
29	Phenotypic RFI, kg/d	23	1.91	29.83	2.31	2.75
29	Genetic RFI, kg/d	23	1.89	29.59	2.34	2.81

[a] QTL effects with F-values greater than chromosome-wise threshold ($P = 0.10$, $P = 0.05$, and $P = 0.01$) are shown.

[b] From 2000 permutations.

Table 8.3. Chromosome-wise significant effects from within-family analysis for daily DMI

BTA	Family	Position, cM	F-value[a]	Threshold F[b] ($P = 0.05$)	Threshold F[b] ($P = 0.01$)	QTL effect, kg/d	SE
1	KA51	11	16.25	12.05	25.06	2.67	0.66
2	KA135	4	11.15	8.81	12.35	-1.23	0.37
3	CB221	15	18.83	9.89	16.50	-2.61	0.60
3	KA135	31	9.27	8.09	12.89	1.98	0.64
3	KA133	121	10.25	6.84	10.78	-1.28	0.40
4	CB221	104	18.18	13.64	23.58	-4.41	1.03
6	KA232	7	12.93	7.48	13.68	-1.45	0.40
8	CB231	2	22.67	13.99	19.80	3.49	0.73
8	KCH41	120	9.07	7.97	11.42	1.33	0.44
10	CB305	97	13.61	9.15	12.86	1.97	0.53
10	KA59	50	12.84	10.33	13.06	-6.72	1.88
10	KA149	82	7.31	6.87	11.00	1.19	0.44
10	KCH28	63	9.05	7.68	10.54	2.24	0.74
11	KA281	124	7.31	7.30	10.85	1.35	0.50
12	KA149	79	11.93	7.18	10.56	-1.22	0.35
14	KA149	105	11.82	7.78	11.34	1.32	0.38
15	CB305	106	15.87	10.30	16.34	2.14	0.54
15	KA149	111	9.70	7.42	11.48	-1.08	0.35
15	KA232	46	22.43	10.21	20.03	-1.57	0.33
15	KCH28	47	8.26	7.51	12.80	-1.16	0.40
17	KA133	70	8.68	7.29	11.18	-1.07	0.36
17	KCH6	9	8.19	7.03	11.96	1.80	0.63
18	CB231	82	18.12	13.03	20.25	3.72	0.87
19	KCH28	41	9.50	7.76	13.03	-1.44	0.47
20	CB467	25	11.25	8.35	14.66	1.78	0.53
20	KA51	72	35.09	9.52	16.39	-3.08	0.52
22	CB231	66	13.86	9.61	14.90	2.88	0.77
22	KA379	12	7.03	7.42	13.13	1.69	0.64
25	CB305	40	8.28	7.98	13.53	-2.08	0.72
27	KA51	40	6.73	6.46	15.13	1.73	0.67
28	KCH28	36	15.48	9.80	14.98	-1.34	0.34
29	KA149	22	12.85	6.01	10.73	-1.22	0.34

[a] QTL effects with QTL effects with F-values greater than chromosome-wise threshold ($P = 0.05$ and $P = 0.01$) are shown; [b] From 2500 permutations.

Table 8.4. Chromosome-wise significant effects from within-family analysis for feed conversion ratio

BTA	Family	Position, cM	F-value[a]	Threshold F[b] ($P = 0.05$)	Threshold F[b] ($P = 0.01$)	QTL effect, kg DM/kg gain	SE
1	KA321	166	22.53	12.21	20.18	-2.44	0.51
2	CB221	91	17.25	11.21	16.40	-5.43	1.31
4	KA149	159	12.00	8.54	13.12	-2.16	0.62
4	KA281	31	8.69	8.16	14.09	-1.89	0.64
5	CB307	47	12.34	10.86	18.22	1.23	0.35
5	KA281	76	12.85	12.69	18.26	2.24	0.62
6	KA133	76	11.67	6.71	10.48	0.88	0.26
6	KCH41	71	7.76	7.25	11.30	-1.15	0.41
8	KA149	109	8.89	8.78	13.08	-1.59	0.53
10	CB231	104	22.65	13.00	26.42	-1.90	0.40
10	KA59	104	15.83	13.02	18.93	0.92	0.23
11	KCH28	4	13.69	9.06	14.74	2.87	0.77
14	KA149	53	9.27	7.13	10.80	-1.05	0.34
15	CB467	59	7.18	7.10	12.66	1.08	0.40
15	KA232	115	12.44	9.72	17.54	2.09	0.59
15	KA379	8	9.41	9.32	21.36	-2.06	0.67
20	KCH28	73	8.47	8.27	13.77	1.35	0.46
23	KA51	34	9.23	10.14	17.50	-0.84	0.28
23	KA135	59	14.03	8.56	12.21	-1.13	0.30
23	KCH28	2	6.79	6.78	10.49	-1.31	0.50
25	CB231	56	11.62	8.38	20.85	-2.01	0.59
26	KA59	56	14.15	10.63	18.49	2.17	0.58
27	CB305	58	11.10	10.65	19.31	1.42	0.43
27	KCH41	38	6.39	5.58	8.53	0.76	0.30
28	KA135	0	10.08	6.92	10.99	1.01	0.32
28	KA321	51	15.26	9.90	15.99	1.88	0.48
28	KCH28	0	26.96	11.46	22.68	3.74	0.72
29	KA379	2	8.78	8.45	15.51	1.90	0.64

[a] QTL effects with QTL effects with F-values greater than chromosome-wise threshold ($P = 0.05$ and $P = 0.01$) are shown; [b] From 2500 permutations.

Table 8.5. Chromosome-wise significant effects from within-family analysis for phenotypic RFI

BTA	Family	Position, cM	F-value[a]	Threshold F[b] ($P = 0.05$)	Threshold F[b] ($P = 0.01$)	QTL effect, kg DM/d	SE
2	CB221	91	17.73	11.82	16.17	-3.57	0.85
2	KA133	11	7.86	7.83	14.72	0.80	0.29
3	CB305	86	10.85	12.16	17.05	-1.36	0.41
10	CB231	100	21.97	13.60	20.76	-2.59	0.55
10	CB305	38	24.87	16.37	40.58	0.79	0.16
10	KA232	30	12.93	10.25	16.55	-2.05	0.57
10	KCH28	65	10.33	7.31	10.32	1.73	0.54
12	KA149	78	9.59	7.19	11.45	-0.67	0.22
14	KCH6	39	14.96	8.06	12.08	2.05	0.53
15	CB305	107	11.44	11.29	16.99	1.21	0.36
15	KCH41	41	7.01	6.68	10.84	0.51	0.19
17	KA133	76	7.55	7.39	10.85	-0.75	0.27
19	KCH28	66	23.01	7.44	12.89	-1.54	0.32
20	CB467	24	16.42	8.42	15.27	1.37	0.34
20	KA51	73	28.74	9.10	16.44	-2.12	0.40
20	KA133	58	9.85	7.05	10.38	1.23	0.39
20	KA149	41	6.08	5.45	8.33	0.61	0.25
23	KA59	14	19.74	9.58	30.19	1.03	0.23
24	CB221	24	10.10	7.84	12.61	1.98	0.62
24	KA321	6	14.48	9.71	16.79	1.88	0.49
25	CB305	38	11.39	8.04	15.90	-1.43	0.42
26	CB305	40	8.24	7.87	12.81	1.08	0.38
29	CB221	47	10.00	8.46	14.20	-2.34	0.74
29	CB231	22	12.45	11.33	20.62	6.26	1.77
29	KA149	22	6.07	5.57	8.25	-0.55	0.23

[a] QTL effects with QTL effects with F-values greater than chromosome-wise threshold ($P = 0.05$ and $P = 0.01$) are shown;

[b] From 2500 permutations.

Table 8.6. Chromosome-wise significant effects from within-family analysis for genetic RFI

BTA	Family	Position, cM	F-value[a]	Threshold F[b] ($P = 0.05$)	Threshold F[b] ($P = 0.01$)	QTL effect, kg DM/d	SE
2	CB221	91	24.88	13.20	18.49	-4.26	0.85
2	KA133	11	8.95	7.55	11.64	0.85	0.28
2	KA232	122	9.69	9.31	14.98	0.94	0.31
3	CB305	86	13.19	11.37	18.03	-1.50	0.41
10	CB231	101	22.78	13.34	26.04	-2.53	0.53
10	KA59	59	12.57	12.47	51.77	1.07	0.30
10	KA232	29	10.54	10.15	15.92	-2.05	0.63
10	KCH28	65	13.81	7.72	12.46	2.14	0.58
14	KCH6	40	13.27	8.09	12.62	2.09	0.57
15	KA379	8	9.75	8.96	17.48	-1.18	0.38
15	KCH41	40	7.23	7.23	12.21	0.52	0.19
17	KA133	76	7.34	7.32	13.67	-0.73	0.27
18	KA135	6	6.75	6.55	10.32	0.75	0.29
19	KCH28	69	18.81	6.87	10.70	-1.62	0.37
20	CB467	23	12.88	8.04	13.12	1.26	0.35
20	KA51	74	23.31	9.17	16.25	-1.93	0.40
20	KA133	58	9.42	7.08	12.02	1.21	0.39
20	KA149	44	6.18	5.55	9.22	0.64	0.26
22	KA149	40	6.87	5.39	9.29	0.67	0.25
24	CB221	24	8.62	8.01	12.65	2.18	0.74
24	KA321	6	14.58	8.36	11.94	1.90	0.50
25	CB305	38	11.13	10.37	15.94	-1.48	0.44
26	CB305	40	9.35	8.84	13.42	1.17	0.38
26	KA59	57	17.63	11.31	36.98	2.30	0.55
29	CB221	49	9.27	9.10	17.05	-2.73	0.90
29	CB231	22	12.05	11.83	21.82	6.08	1.75

[a] QTL effects with QTL effects with F-values greater than chromosome-wise threshold ($P = 0.05$ and $P = 0.01$) are shown;

[b] From 2500 permutations.

Table 8.7. Chromosome-wise significant effects from within-family analysis for partial efficiency of growth

BTA	Family	Position, cM	F-value[a]	Threshold F[b] ($P = 0.05$)	Threshold F[b] ($P = 0.01$)	QTL effect	SE
1	KA51	7	18.63	11.84	19.40	-0.330	0.080
2	CB221	91	65.72	21.26	34.79	0.188	0.023
2	KA133	12	8.50	8.45	13.01	-0.045	0.015
2	KA232	121	9.27	8.81	12.42	-0.047	0.015
3	CB305	86	12.05	10.19	18.63	0.079	0.023
10	CB231	100	12.31	11.65	19.27	0.151	0.043
10	CB305	40	17.78	19.12	43.33	-0.035	0.008
10	KA59	58	19.23	14.64	27.68	-0.057	0.013
10	KCH28	64	17.80	7.51	10.65	-0.115	0.027
14	KCH6	41	20.24	7.79	11.50	-0.161	0.036
15	KA379	8	13.35	9.93	15.89	0.078	0.021
17	KA321	83	10.83	7.90	12.21	-0.074	0.023
19	KCH28	90	12.59	6.79	10.45	0.073	0.021
19	KCH41	35	7.70	7.06	10.89	-0.043	0.015
20	KA51	73	13.34	10.39	18.23	0.101	0.028
20	KA133	58	8.00	6.86	10.35	-0.059	0.021
23	KA135	59	13.11	9.09	16.59	0.047	0.013
25	CB305	39	8.98	8.55	13.66	0.076	0.025
26	CB305	40	14.78	8.75	16.10	-0.072	0.019

[a] QTL effects with QTL effects with F-values greater than chromosome-wise threshold ($P = 0.05$ and $P = 0.01$) are shown; [b] From 2500 permutations.

Table 8.8. Chromosome-wise significant effects from within-family analysis for daily feeding duration

BTA	Family	Position, cM	F-value[a]	Threshold F[b] ($P = 0.05$)	Threshold F[b] ($P = 0.01$)	QTL effect, min/d	SE
3	CB305	84	15.38	10.53	19.19	-14.76	3.76
5	KCH28	6	14.50	10.27	17.26	-18.23	4.79
11	KCH28	109	12.61	6.87	10.47	-50.73	14.29
12	KCH28	85	14.54	7.51	13.84	20.42	5.36
14	KCH6	71	12.52	8.38	13.08	16.60	4.69
14	KA135	107	9.07	6.93	10.52	12.33	4.10
14	CB467	63	9.42	8.70	14.54	-30.75	10.02
16	KA232	46	7.59	7.33	11.55	-22.66	8.23
17	KCH28	31	19.98	9.68	17.45	-35.06	7.84
18	KA51	6	10.49	9.96	18.90	-18.96	5.85
19	KCH28	26	8.97	7.63	12.92	-25.70	8.58
21	KA133	29	7.63	6.25	9.75	14.27	5.17
22	KA321	94	9.74	9.40	17.78	34.77	11.14
22	KA379	12	12.32	6.63	10.48	20.52	5.85
23	CB467	46	8.20	7.10	12.63	-23.34	8.15
23	KA135	2	8.67	8.51	13.95	-11.73	3.98
26	CB467	16	6.93	6.30	10.86	-20.00	7.60
26	KA232	31	7.43	6.08	11.70	-26.13	9.58
28	KCH28	27	16.52	6.25	15.98	-25.41	6.25

[a] QTL effects with QTL effects with F-values greater than chromosome-wise threshold ($P = 0.05$ and $P = 0.01$) are shown; [b] From 2500 permutations.

Table 8.9. Chromosome-wise significant effects from within-family analysis for daily feeding head down time

BTA	Family	Position, cM	F-value[a]	Threshold F[b] ($P = 0.05$)	Threshold F[b] ($P = 0.01$)	QTL effect, min/d	SE
1	KA135	165	10.20	6.83	11.68	12.73	3.99
2	KA135	4	11.11	8.17	12.66	-10.66	3.20
2	KA51	15	19.66	11.51	23.61	-16.45	3.71
3	CB231	66	12.14	11.99	30.07	24.67	7.08
3	KCH6	8	13.62	6.90	10.91	-14.54	3.94
5	KCH28	6	19.54	11.09	15.48	-16.21	3.67
5	KCH41	60	15.48	9.13	12.74	9.46	2.40
10	CB231	91	19.36	14.33	36.63	-21.93	4.98
12	CB305	51	11.72	9.97	16.06	15.78	4.61
12	KA59	12	27.24	12.01	25.15	-26.05	4.99
12	KCH28	84	13.95	8.36	12.79	16.30	4.36
14	CB467	58	18.70	9.11	14.72	-26.16	6.05
15	CB305	80	11.11	11.81	18.32	-18.97	5.69
15	KA133	38	9.10	7.21	10.50	10.17	3.37
16	KA232	46	7.63	7.15	13.67	-13.64	4.94
17	CB221	15	10.43	9.45	13.01	-9.12	2.82
17	KA133	71	11.81	7.31	12.10	-11.80	3.43
17	KA149	2	9.54	6.55	9.73	-4.47	1.45
17	KCH28	34	11.05	8.74	13.32	-27.14	8.17
18	KCH28	30	9.69	9.57	14.97	-12.65	4.07
20	KCH6	44	7.95	6.93	11.15	16.72	5.93
22	KA379	12	7.29	6.42	11.95	15.22	5.63
23	KA59	14	22.13	8.14	24.48	21.39	4.55
26	CB467	15	7.11	5.87	10.88	-13.07	4.90
26	KA232	20	10.93	6.78	11.19	-22.00	6.65
28	KA379	0	8.36	8.27	18.10	14.77	5.11

[a] QTL effects with QTL effects with F-values greater than chromosome-wise threshold ($P = 0.05$ and $P = 0.01$) are shown;

[b] From 2500 permutations.

Table 8.10. Chromosome-wise significant effects from within-family analysis for daily feeding frequency

BTA	Family	Position, cM	F-value[a]	Threshold F[b] (*P* = 0.05)	Threshold F[b] (*P* = 0.01)	QTL effect, events/d	SE
2	KA51	133	13.51	10.91	17.97	9.34	2.54
4	CB221	124	19.66	13.38	32.82	6.54	1.47
4	KA149	128	10.47	8.49	13.36	11.20	3.46
6	KA133	83	8.10	7.28	11.19	4.38	1.54
7	KA51	119	20.83	10.77	21.65	9.83	2.15
7	KA379	131	9.15	8.91	14.20	5.05	1.67
8	KCH28	85	7.32	7.28	11.18	7.13	2.63
10	CB231	32	36.28	23.80	72.30	-31.49	5.23
10	CB305	54	11.15	9.26	16.12	-9.74	2.92
11	KA51	70	11.33	11.00	23.66	8.49	2.52
12	KCH41	50	6.88	6.79	10.22	4.96	1.89
14	CB221	70	17.45	13.47	22.88	4.45	1.07
16	CB349	39	77.96	31.80	95.52	7.63	0.86
18	KA321	45	12.89	12.81	25.80	-12.20	3.40
19	KCH41	29	8.10	6.49	10.10	6.87	2.41
20	KA51	58	11.30	9.06	17.17	-8.74	2.60
22	KA379	21	7.88	7.21	11.42	5.85	2.08
23	CB305	51	16.26	11.17	17.23	-16.73	4.15
23	KCH28	40	10.05	8.51	11.34	-9.51	3.00
28	KA59	18	25.99	14.54	22.84	-18.29	3.59
28	KCH28	55	10.41[c]	11.34	16.49	15.82	4.90

[a] QTL effects with QTL effects with F-values greater than chromosome-wise threshold (*P* = 0.05 and *P* = 0.01) are shown;

[b] From 2500 permutations.

Table 8.11.Chromosome-wise significant effects from within-family analysis for flight speed

BTA	Family	Position, cM	F-value[a]	Threshold F[b] (*P* = 0.05)	Threshold F[b] (*P* = 0.01)	QTL effect, m/s	SE
4	KA133	56	16.85	8.12	10.81	-0.27	0.07
4	KA379	61	12.92	9.62	16.05	-1.09	0.47
8	KA379	105	13.86	10.41	17.74	0.57	0.15
12	CB467	53	7.45	7.19	11.18	0.31	0.11
14	CB467	81	9.99	8.07	12.95	0.49	0.16
15	KCH6	73	10.80	8.58	12.71	0.16	0.05
23	KA281	0	11.72	9.79	15.26	-0.22	0.07
23	KA379	88	7.54	7.52	13.20	-0.51	0.19
24	KA321	64	11.15	9.05	16.99	0.82	0.25
26	KA321	12	8.87	4.96	9.11	-1.93	0.65
27	KA133	34	8.06	6.38	9.85	-0.15	0.05
27	KA232	8	12.45	8.25	15.37	-0.27	0.08
28	KA321	41	15.91	11.36	26.22	-0.86	0.22

[a] QTL effects with QTL effects with F-values greater than chromosome-wise threshold (*P* = 0.05 and *P* = 0.01) are shown

[b] From 2500 permutations.

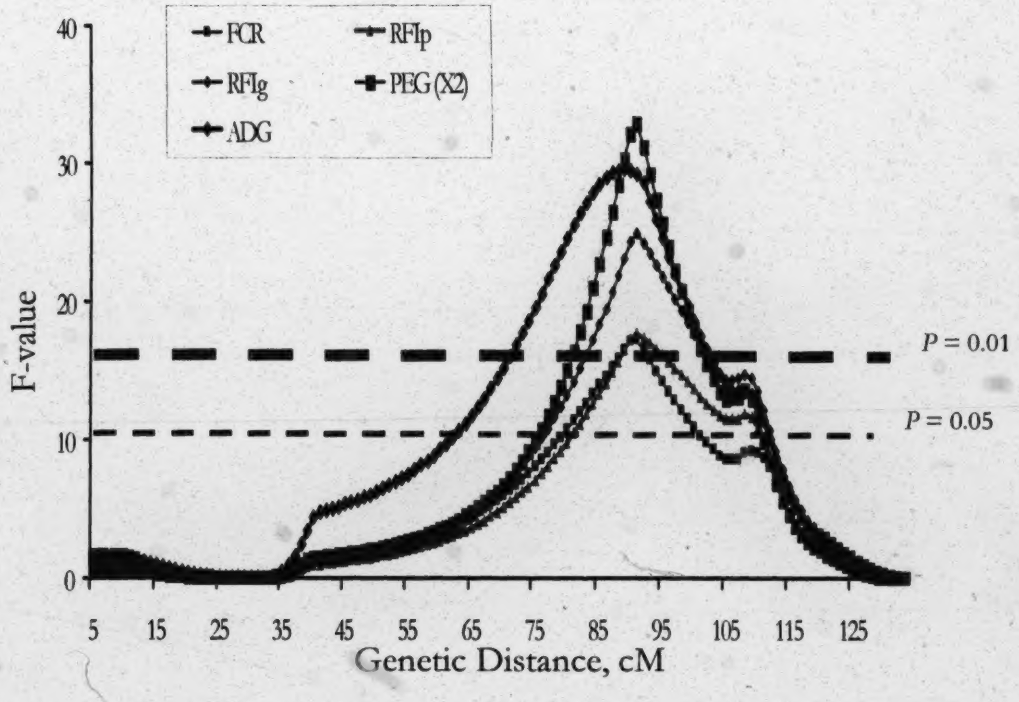

Figure 8.1. Within-family QTL results for growth rate and feed efficiency traits on bovine chromosome 2 in a crossbred composite cattle population.

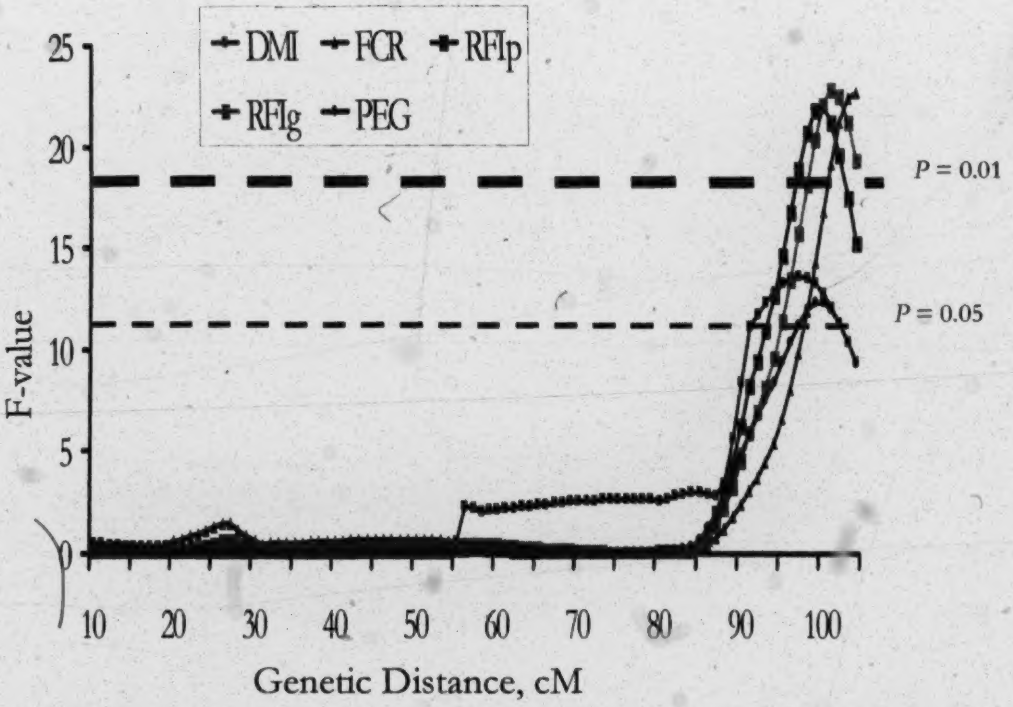

Figure 8.2. Within-family QTL results for DMI, feed conversion ratio (FCR), phenotypic RFI (RFIp), genetic RFI (RFIg) and partial efficiency of growth (PEG) on bovine chromosome 10 in a crossbred composite cattle population.

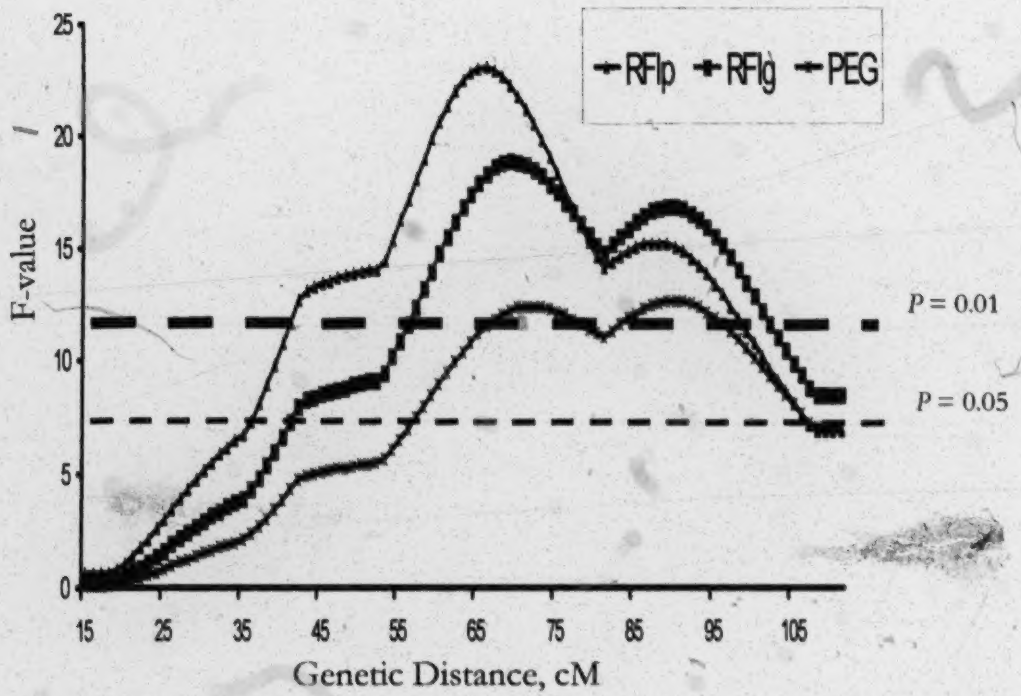

Figure 8.3. Within-family QTL results for phenotypic RFI (RFIp), genetic RFI (RFIg) and partial efficiency of growth (PEG) on bovine chromosome 19 in a crossbred composite cattle population.

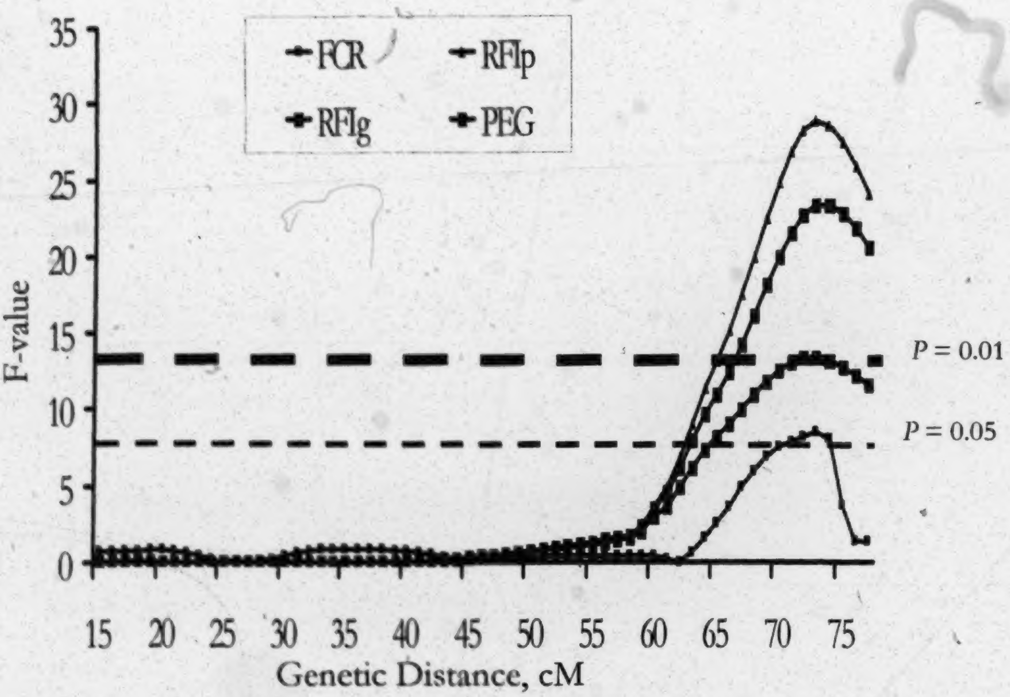

Figure 8.4. Within-family QTL results for feed conversion ratio (FCR), phenotypic RFI (RFIp), genetic RFI (RFIg) and partial efficiency of growth (PEG) on bovine chromosome 20 in a crossbred composite cattle population.

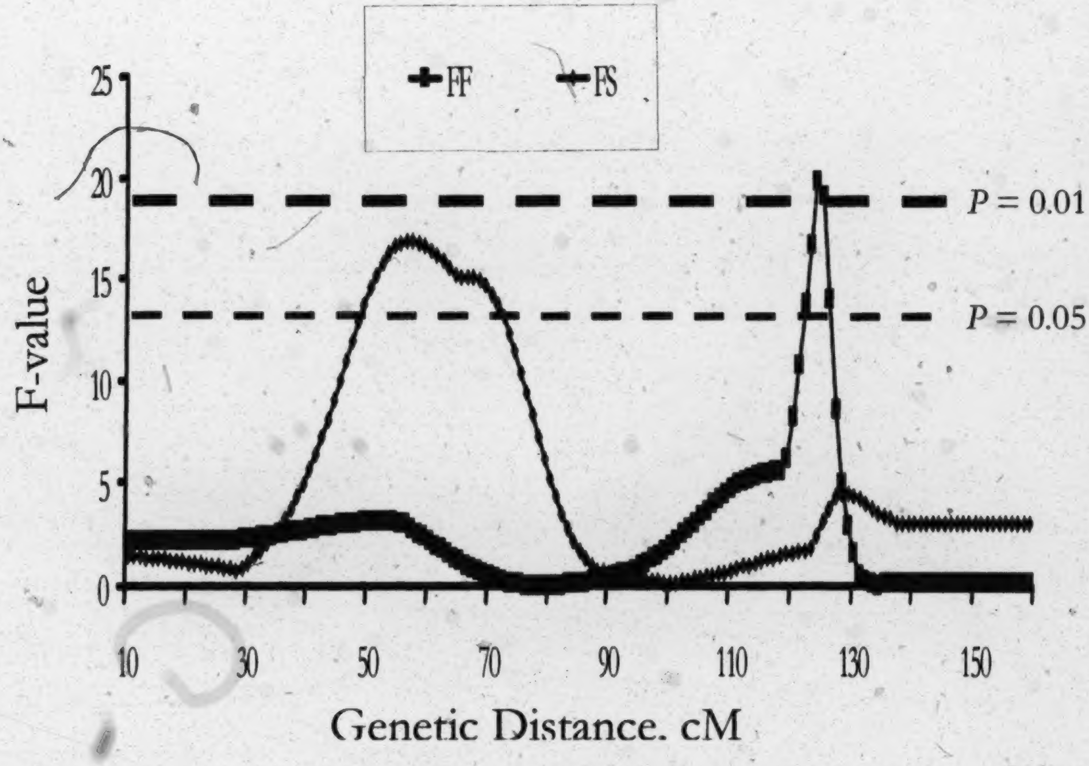

Figure 8.5. Within-family QTL results for daily feeding frequency (FF) and flight speed (FS) on BTA 4 in a crossbred composite cattle population.

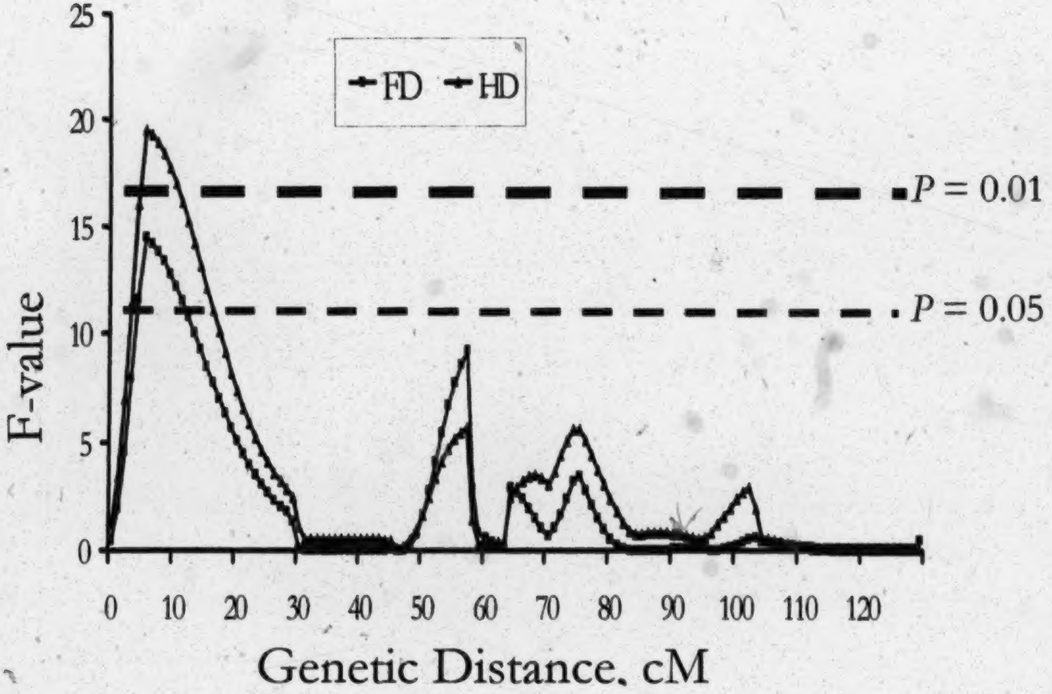

Figure 8.6. Within-family QTL results for daily feeding duration (FD) and feeding head down time (HD) on BTA 5 in a crossbred composite cattle population.

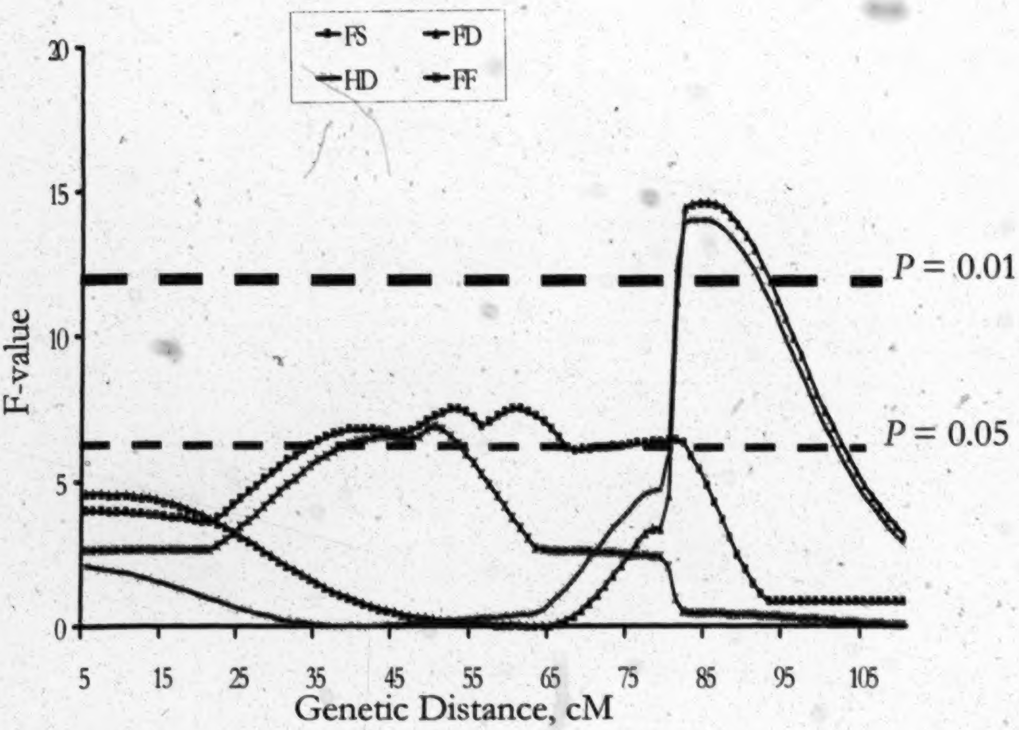

Figure 8.7. Within-family QTL results for daily head down time (HD), feeding duration (FD), feeding frequency (FF), and flight speed (FS) on BTA 12 in a crossbred composite cattle population.

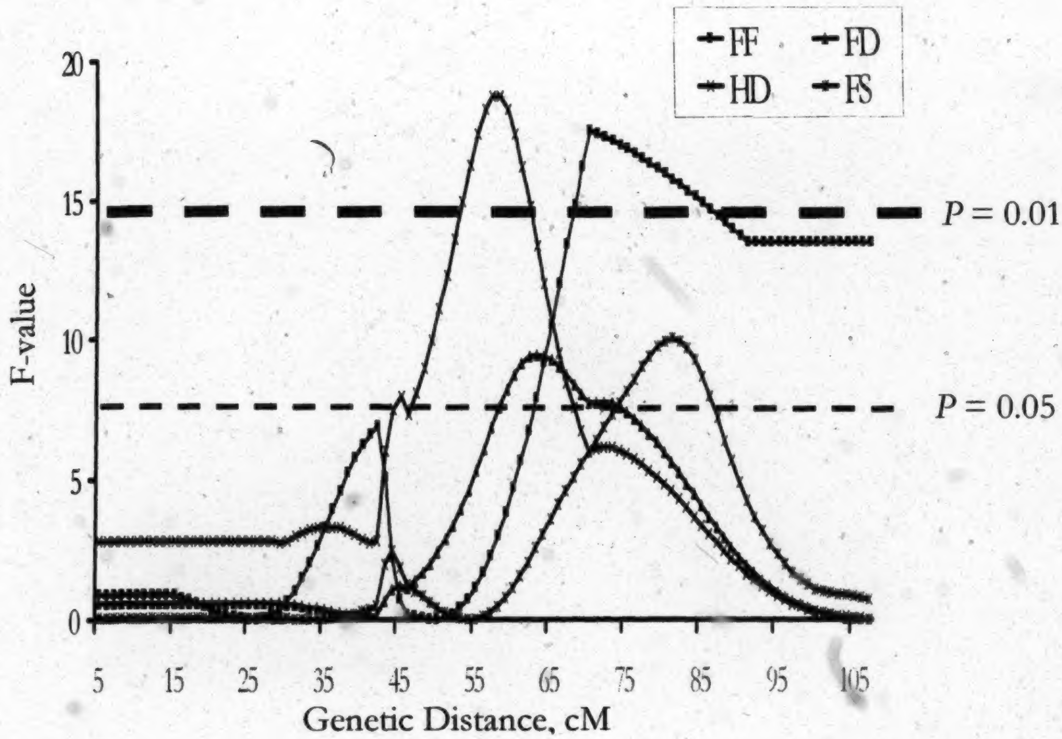

Figure 8.8. Within-family QTL results for daily head down time (**HD**), feeding duration (**FD**), feeding frequency (**FF**), and flight speed (**FS**) on BTA 14 n a crossbred composite cattle population.

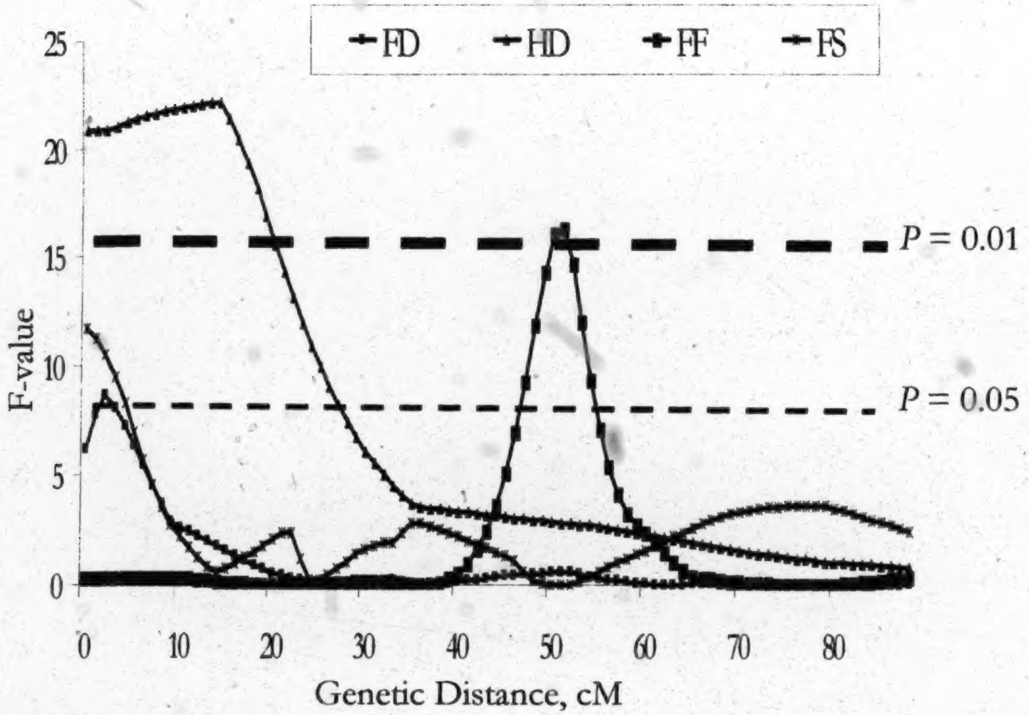

Figure 8.9. Within-family QTL results for daily head down time (**HD**), feeding duration (**FD**), feeding frequency (**FF**), and flight speed (**FS**) on BTA 17 in a crossbred composite cattle population.

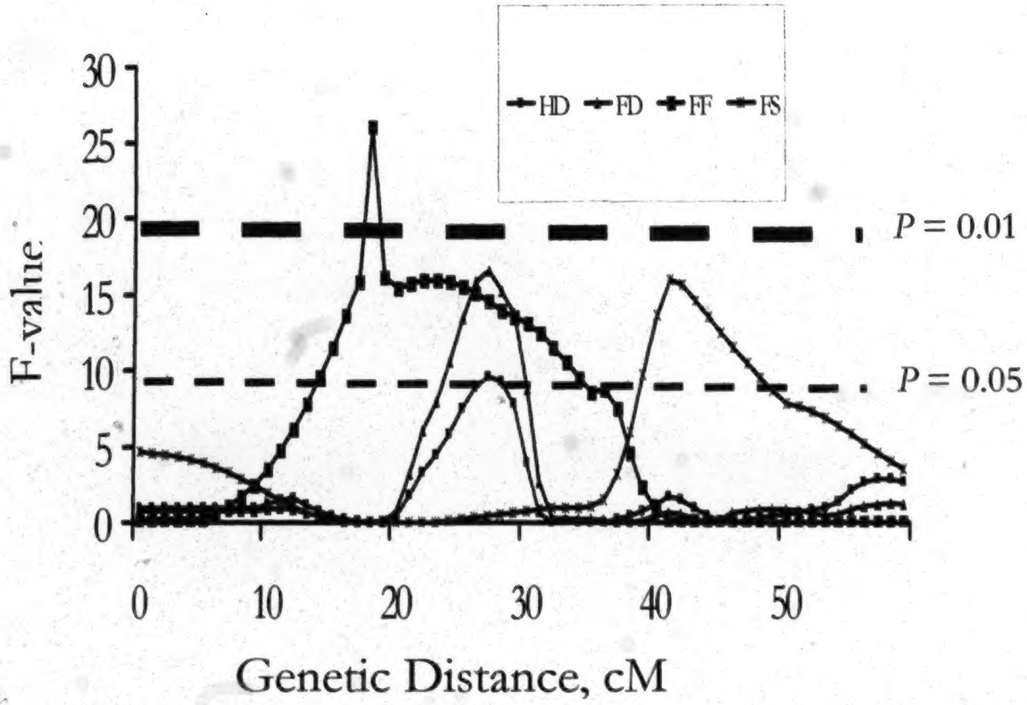

Figure 8.10. Within-family QTL results for daily head down time (**HD**), feeding duration (**FD**), feeding frequency (**FF**), and flight speed (**FS**) on BTA 28 in a crossbred composite cattle population.

CHAPTER 9

General Discussion and Conclusion

9.1 General Discussion

The cost of feeding beef cattle is the single largest variable cost in most beef production systems, accounting for as high as 70% of total variable cost. Feed intake and feed efficiency of beef cattle have therefore been identified as economically relevant traits, which have a large impact on the production cost, profitability, global competitiveness, as well as environmental sustainability of the beef industry. There have been considerable recent research efforts in a number of countries aimed at genetically improving the overall biological and economic efficiency of the total beef production system (as reviewed in Herd et al., 2003; Crews, 2005). Serious consideration is also being given to those characteristics that determine consumer acceptability of the final product. The primary objective of this research project was to characterize the genetic variation in feed intake and feed efficiency of individual crossbred composite cattle from an experimental population and exploit the genetic variation to identify genes influencing feed intake and feed efficiency.

The first study (Chapter 3) examined the genetic and phenotypic relationships among feed intake and different measures of feed efficiency, and evaluated their relationships with growth, ultrasound, and carcass merit of beef cattle. Feed intake and three indices of feed efficiency, namely feed conversion ratio (**FCR**), residual feed intake (**RFI**), and partial efficiency of growth (**PEG**) were considered. The study also compared RFI calculated from phenotypic regression (**RFIp**) and genetic regression (**RFIg**) of ADG and metabolic BW on daily feed intake. The results of this study were consistent with those of previous studies (as reviewed by Archer et al., 1999; Herd et al., 2003; Crews et al., 2005), which indicated that genetic variation in feed efficiency exists in beef cattle. High genetic and phenotypic correlations observed between RFIp and RFIg were consistent with the findings of Hoque et al. (2005), indicating that the two indices are very similar, though in the present study the heritability estimate for the latter (0.42) was higher than the former (0.21). Both indices of RFI were favourably correlated with feed intake, FCR, and PEG.

Phenotypic RFI was independent of ADG and BW, and had low and moderate genetic correlations with the same traits, but the very large standard errors associated with the genetic correlation estimates makes it difficult to determine whether these correlations are indeed different from zero. On the other hand, RFIg was genetically independent of ADG and BW and was phenotypically independent of BW, but had a weak phenotypic correlation with ADG. The genetic and phenotypic correlations of RFIp and RFIg with ultrasound and carcass traits were generally similarly low or not different from zero, except moderate to high genetic correlations observed with ultrasound and carcass LM area and carcass lean meat yield. Other studies have reported associations consistent with the findings of this study (Arthur et al., 2001; Herd and Bishop, 2000; Schenkel et al., 2004), though the correlations observed in these other studies were low. The present results also indicated that, unlike RFI, feed intake and FCR are much more strongly correlated with ADG and BW. Though PEG was similar to RFI in terms of relationships with a lot of the traits considered, it was not genetically and phenotypically independent of ADG.

In Chapter 4, we employed techniques in nutritional energetics (mainly digestibility and indirect calorimetry) to study the potential biological mechanisms contributing to the variation in feed efficiency of beef cattle. The biological reasons underlying the observed variations in feed efficiency, independent of growth and BW were largely unknown, though several proposals have been made (Johnson et al., 2003; Richardson and Herd, 2004). The differences in carcass merit and body composition among animals with different RFI from studies reported thus far account for only a small proportion (generally 5%) of the observed variations in energetic efficiency among these animals. Potentially, differences in other processes such as heat increment of feeding and visceral metabolism, level of feeding activity and feeding behaviour, nutrient turnover, and digestive functions may also contribute to the variations in RFI (Oddy and Herd, 2001; Richardson and Herd, 2004).

The results of this study demonstrated that differences among animals in metabolizability (mainly digestibility and methane production), heat production, and energy retention may be responsible for a major part of the variation among animals in residual feed intake. Methane production differences between efficient and inefficient animals may be related to potential links between host genetics and rumen microorganisms (Hackstein et al., 1996) and the possibility of differences in volatile fatty acid profiles among animals with different RFI (Russell and Gahr, 2000). Part of the variation in efficiency of energy retention may also be attributed, among other factors, to differences in digestibility of the diet and faecal production (Ferrell and Jenkins, 1998), but was less related to energy lost through urine. The significantly higher heat production in high RFI animals may be associated with observed significantly greater visceral organ weights (Ferrell and Jenkins, 1998; Basarab et al., 2003) and the associated high energy requirements (Reynolds, 2002) and protein turnover (Webster, 1980).

The study in Chapter 5 examined the genetic and phenotypic variations in measures of feeding behaviour and temperament and their potential associations with measures of growth, feed intake, feed efficiency, and carcass merit. Direct heritability estimates for the feeding behaviour traits were moderate and consistent with values reported in cattle (Robinson and Oddy, 2004) or sheep (Cammack et al., 2005). The direct heritability estimate for flight speed was high and consistent with those reported by Burrow (2001) for cattle. The genetic and phenotypic correlations of the measures of feeding behaviour with feed intake and feed efficiency indicate that feeding behaviour traits could potentially be used to predict level of intake and feed efficiency in cattle. The results also confirm suggested contributions of feeding behaviour and activity to the observed variations in RFI (Oddy and Herd, 2001; Johnson et al., 2003; Richardson and Herd, 2004). Although temperament had a moderate relationship with feed intake, growth, and carcass merit (Burrow and Dillon 1997; Voisinet et al., 1997; Fox et al., 2004), the potentially moderate genetic correlation with RFI and PEG implies that strategies to make adjustments for temperament may be required in the implementation of selection for RFI and PEG.

Chapter 6 describes a study to evaluate the potential genetic and phenotypic relationships of circulating levels of the appetite regulating hormone leptin with measures of performance, feed intake, feed efficiency, and carcass merit. The study provided experimental evidence indicating that serum leptin concentration is moderately heritable, and can be used to predict carcass merit and body composition (Geary et al., 2003; Minton et al., 1998). Breed differences in serum leptin levels were consistent with differences in body fatness and carcass merit. The results also demonstrated low to moderate genetic correlations between RFI and PEG with serum leptin, though phenotypic correlations were not different from zero. Feed intake was negatively correlated with serum leptin, though the corresponding phenotypic correlation was positive and weak. These results indicated that the relationship between serum leptin concentration and body fatness may be stronger than the relationship of serum leptin with feed intake. This is consistent with Delavaud et al. (2000), who also observed that the relationship between circulating leptin levels and the day-to-day variation in nutritional status in cattle may be very low (17%) when compared to the relationships of circulating leptin with the long term effect on adipose tissue mass (35%).

In Chapter 7 we evaluated the potential of leptin as a candidate gene for growth, feed intake, feed efficiency, and carcass merit. Several single nucleotide polymorphisms (**SNP**) in the leptin gene and its promoter were evaluated for associations with these traits. A previously reported (Buchanan et al., 2002) C – T missense mutation at position 305 in exon 2 of the leptin gene was shown to be associated with higher subcutaneous fat and lower lean meat yield in a commercial population of cattle (Nkrumah et al., 2004). This mutation results in an arginine (R; CGC) to cystein (C; TGC) substitution at amino acid 4 (R4C) in the leptin molecule resulting in a putative conformational change. Subsequent analysis of this mutation using the data from the experimental population in the present study confirmed the association with backfat thickness and carcass lean. No significant associations with measures of performance, feed efficiency or the remaining traits were observed for this mutation. These associations have been confirmed by some studies (Schenkel et al., 2005; Woodward et al., 2005) but not by others (Crews et al., 2004; Lagonigro et al., 2003).

Two polymorphisms, UASMS2 (C-T substitution) and UASMS3 (G-C substitution) located at nucleotide positions 528 and 1759 in the leptin promoter (GenBank accession no. AB070368) were previously reported (Nkrumah et al., 2005) to be associated with serum leptin concentration, growth, feed intake, and carcass merit in a subset of the experimental population data of this study. Strong interests in the leptin promoter mutations stem from the potential association of such polymorphisms with the sequence elements and factors regulating leptin gene expression. Subsequent analysis of the above promoter polymorphisms using the complete experimental dataset indicated that the associations with UASMS3 may be due to its significant linkage disequilibrium with UASMS2. The UASMS2 SNP was shown to be associated with serum leptin concentration, feed intake, and carcass merit. There were also trends of associations with residual feed intake. Some of the above associations with UASMS2 have been confirmed (Crews et al., 2004; Woodward et al., 2005). A recent study by Schenkel et al. (2005) did not find any of the associations with carcass merit reported for UASMS2.

In addition to the above SNP, a C-T mutation at position 321 (GenBank accession no. BTA512639; EMBL Accession no. AJ512639) in exon 3 of the leptin gene was evaluated. The SNP results in an alanine (A; GCG) to valine (V; GTG) substitution at amino acid 59 (A59V) in the β-helix region that is conserved between species in the leptin molecule. The A59V SNP has previously been shown to be associated with serum leptin concentration in pregnant dairy cows (Liefers et al., 2003). The present study observed associations between A59V and serum leptin concentration, growth rate, feed conversion ratio, relative growth rate, and Kleiber ratio. The A59V SNP also showed associations with ultrasound and carcass merit. We subsequently evaluated genotype combinations of UASMS2 and A59V for their associations with the traits considered using haplotype regression analysis. This analysis showed much stronger associations of the two SNP with serum leptin, ADG, relative growth rate, Kleiber ratio, and measures of ultrasound and carcass merit. Further confirmation studies involving these new associations need to be carried out before any applications to marker-assisted selection.

In Chapter 8, we report a *Bos taurus* autosomal (**BTA**) genome scan with 455 genetic markers across 16 paternal half-sib families to identify quantitative trait loci (**QTL**) for feed intake, feed efficiency, feeding behaviour, and temperament in beef cattle. Fifty-one highly significant (chromosome-wise $P < 0.01$) QTL effects were identified within families, and 22 significant (chromosome-wise $P < 0.05$) QTL effects were detected across families. A total of 4176 trait–family–chromosome hypothesis tests were carried out within families with 42 expected by chance at chromosome-wise $P = 0.01$, and 51 were detected. For the across-family analysis, a total of 261 trait–chromosome hypothesis tests were carried out with 13 expected by chance at chromosome-wise $P = 0.05$, and 22 were detected. It is important to note that the very high genetic and phenotypic correlations among the traits tested in this study imply that the different multiple hypothesis tests carried out are generally not independent. A highly stringent procedure for correcting for multiple testing (such as mentioned above) is therefore not appropriate for determining Type I error rates for the hypothesis tests carried out in this study (Perneger, 1998).

Generally, more QTL were detected within families than across families because not all families are informative (heterozygous) for a detected QTL. Highly significant (chromosome-wise $P < 0.01$) QTL affecting daily DMI were found on BTA 3, 8, 10, 12, 15, 20, 28, and 29. For FCR, highly significant QTL were detected on BTA 1, 2, 6, 23, and 28. The strongest (most significant) QTL for RFI were detected on BTA 2, 10, 14, 19, 20, and 29. Highly significant PEG QTL were detected on BTA 2, 10, 14, and 19. For the feeding behaviour traits, highly significant QTL were detected on BTA 11, 12, 17, 22, and 28 for daily feeding duration; BTA 3, 5, 12, and 14 for daily feeding head down time; and BTA 28 for daily feeding frequency. For flight speed, a highly significant QTL was detected on BTA 4.

9.2 General conclusions and recommendations

The results of this study, as well as other published evidence, indicate that there is considerable genetic and phenotypic variation in beef cattle feed efficiency. The direct heritability estimates for several feed efficiency indices are similar to those reported for growth rate and body weight. The high cost of feeding beef cattle implies that, genetic improvement of feed efficiency complemented with appropriate adjustments for animal behaviour and temperament is the logically appropriate approach to make the beef industry as competitive as the poultry and swine industries. The incorporation of feed efficiency traits into classical genetic evaluation programs, though a highly commendable idea, represents considerable costs to the beef industry due to the expense involved in the recording of feed intake of individual cattle. The application of genomics-based feed efficiency technology on the other hand, represents a very economically viable option.

Several QTLs underlying feed intake and feed efficiency have been identified in the present study. Further efforts aimed at fine-mapping these QTLs alongside the analysis of specific candidate genes will lead to the identification of the causative mutations underlying the genetic variation in feed efficiency. The present study considered leptin as a candidate for feed intake and feed efficiency. However, the results indicated stronger associations of bovine leptin polymorphisms with body fatness rather than residual feed intake. Haplotypes constructed between one mutation in the leptin promoter and a mutation in leptin exon 3 showed associations with feed conversion ratio, relative growth rate, and Kleiber ratio, but not with residual feed intake. Evolving evidence regarding the mechanisms involved in the regulation of feed intake and energy homeostasis in mammals point towards a complex redundant system that functions under normal conditions to minimize the effects of short-term fluctuations in energy intake and expenditure. Thus, polymorphisms in other neuropeptides and neurotransmitters that have been shown to be involved in the homeostatic mechanism for regulation of feed intake and energy balance in mammals should be carefully evaluated. Additional potential candidate genes may be identified through fine-mapping of the QTLs for feed efficiency and analysis of gene expression using DNA microarrays on animals with extreme feed efficiency phenotypes.

A lot is to be gained in the application of genomic information for feed efficiency traits to guide selection decisions. Recent proposals regarding how genomic information should be employed in genetic improvement have suggested a complementation of existing classical quantitative genetic evaluation approaches. While this may be a very conservative approach to safeguard against potential negative effects of false-positive genomic results, it harbours the danger of being counter-productive in that it can overshadow and slow down the potential progress that is to be made from marker-assisted selection. The logical approach will be to exploit genomics-based feed efficiency technology to its full potential, as long as results of association studies properly replicated and revaluated in other independent studies.

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APPENDICES

APPENDIX ONE

Calculation of Residual Feed Intake

Residual feed intake (RFI) relates to the variation in feed consumption between animals beyond that related to differences in growth rate and BW. It is defined as the difference between an animal's actual feed intake and its expected feed intake based on its BW and growth rate over a specified period. Mathematically, **RFI = Actual DMI – Expected DMI.** Each animal's actual DMI is calculated from feed intake data obtained from the Growsafe feed intake recording system. In the present study, data from a 70 d test was used.

Actual DMI is often expressed as standardized DMI (**STDMI**), which is the intake of the animal standardized to a dietary energy density of 10 Mj ME/kg. To do this, the total feed intake of the animal (as fed) is multiplied by the DM content of the diet to obtain the total DMI of each animal over the test period. This value is then multiplied by the ME content of the diet and divided by 10 to obtain the standardized DMI. The ME content of the diet can be estimated from NRC tables based on the individual dietary ingredients or from a digestibility trial followed by adiabatic bomb calorimetry. The total STDMI of each animal is then divided by the test duration to obtain the average daily STDMI (i.e. daily DMI, kg/d) of each animal.

Expected DMI is predicted from NRC equations based on the average metabolic body size (**MWT, kg$^{0.75}$**) and ADG (kg/d) of the animal on test. Generally, Expected DMI has been calculated from Phenotypic Regression (Phenotypic RFI, **RFIp**) of daily DMI on MWT and ADG. ADG is obtained as the coefficient of the linear regression of BW measurements over time, and MWT is calculated as the regression intercept (start weight) plus ADG times half of the total test duration.

Thus, RFIp was calculated as: **RFIp = DMI – β_g x ADG – βw x MWT**, where

$$\begin{bmatrix} \beta_g \\ \beta_w \end{bmatrix} = P^{-1}C$$

Where, ***P*** = phenotypic variance of MWT, phenotypic covariance of ADG and MWT and phenotypic variance of ADG; ***C*** = phenotypic covariances of MWT and ADG with DMI. The phenotypic regression coefficients can easily be obtained with standardized software such as MS Excel or SAS.

Kennedy et al. (1993) argued that phenotypic RFI may show genetic correlations with its component traits, and proposed that RFI may be calculated from genetic regression (genetic RFI, RFIg). Subsequently, Hoque and Oikawa (2004) and Crews (2005) showed that RFIg may be calculated from genetic regression as follows: **RFIg = DMI –** $\boldsymbol{\beta_g}$ **x ADG –** $\boldsymbol{\beta w}$ **x MWT**, where

$$\begin{bmatrix} \beta_g \\ \beta_w \end{bmatrix} = G^{-1}C$$

G = genetic variance of MWT, genetic covariance of ADG and MWT, and genetic variance of ADG; ***C*** = genetic covariance of MWT and ADG with DMI. Genetic (co) variances are obtained from software such as ASREML (Gilmore et al., 2000) using an mixed animal model as described in the thesis.

It may be appropriate to correct feed intake and BW measurements from different contemporary groups for all systematic effects.

APPENDIX TWO

Protocol for high-salt phenonol/chloroform DNA Extraction.

DNA extraction from blood was carried out using a high salt phenol/chloroform extraction method. The following standard solutions are required for DNA extraction

1. **1M KCl** (MW = 74.56g/mole): Dissolve 7.456g KCl in ~100ml H_2O (fill to 100mL); Mix solution on stir plate ad autoclave before use.
2. **1M NH_4Cl** (MW = 53.49g/mole): Dissolve 53.49g NH_4Cl in ~1000ml H_2O (fill to 1000mL). Mix solution on stir plate ad autoclave before use.
3. **1x TE:** Combine 10mL 1M Tris (pH 8.0) with 2mL 0.5M Na_2EDTA and 800mL sterilized H_2O. Adjust volume to 1 L with sterile H_2O.
4. **Phenol : Chloroform : isoamyl alcohol (25:24:1):** For a 500 mL solution, Add 250 mL buffer saturated phenol (pH 7.49-7.79) with 250 mL chloroform (24:1, chloroform:isopentyl alcohol). Prepare solution in a fume hood and cover with tinfoil for storage at 4^{O}C).
5. **Chloroform : isoamyl alcohol (24:1)**: For a 500 mL chloroform, add 480 mL chloroform with 20 mL isoamyl alcohol (also called isopentyl alcohol). Prepare solution in a fume hood and cover with tinfoil for storage at room temperature
6. **Lysis Buffer**: Mix 10 mL1M KCl, 150 mL 1M NH_4Cl, 200uL 0.5M EDTA (pH 8.0), and ~840 mL sterilized H_2O (fill to 1000ml).
7. **1M Tris, pH 7.4** (hydroxymethylaminomethane, MW = 121.14g/mole,): Mix. Dissolve 121.14g Tris in 800 mL milli-Q H_2O. Adjust pH to 7.4 by adding ~70 mL concentrated HCl. Adjust volume to 1L with water. Mix solution on stir plate ad autoclave before use.
8. **0.5M Na_4EDTA-$2H_2O$, pH 8.0** (tetrasodium ethyldiaminetetraacetate MW = 416.20g/mole. Dissolve 104.05g Na_4EDTA-2 H_2O 300mL milli-Q H_2O. Stir vigorously on a magnetic stirrer. Adjust pH to 8.0 by adding concentrated HCl. Adjust volume to 1 L with milli-Q H_2O. Mix solution on stir plate ad autoclave before use.
9. **5M NaCl** (NaCl, MW = 58.44g/mole): Dissolve 292.2g NaCl in 800ml milli-Q H_2 H_2O. Adjust volume to 1 L with milli-Q H_2O. Mix solution on stir plate ad autoclave before use.

10. **TBS (Tris Buffered Saline)**: Add 140 mL 1M NaCl to 500 uL 1M KCl, and 250 uL 1M Tris (pH 7.4). Mix with ~860 mL sterilized H_2O.

The following protocol was used to extract DNA from blood.

1. Thaw ~5mL blood sample (preferably in a 50ml tube) overnight at 4°C
2. Add ~12.5 mL cold Lysis buffer solution – fill to 17.5ml mark and incubate on ice for 10 min.
3. Centrifuge at 4000 rpm at 4°C for 20 minutes; remove and discard the supernatant.
4. Add cold 5mL TBS solution cap well and mix gently; centrifuge at 1000 rpm, 4°C for 10 min; remove supernatant. Repeat this step with another 5 mL TBS solution.
5. Remove and discard the supernatant.
6. Add 3 mL TE solution and vortex gently to re-suspend pellet (high speed)
7. Add 200 uL of 0.5M EDTA (pH 8.0), 15 uL of 20mg/mL proteinase-K and 200 uL 10% SDS and incubate at 37 °C overnight while continuously mixing in a shaker.
8. Add 1 mL of 5 M NaCl, 5 mL phenol/chloroform and mix for 20 minutes at room temperature.
9. Centrifuge at 4000 rpm at 4°C for 20 minutes and collect and keep the supernatant
10. Add 5 mL of chloroform:isoamyl alcohol solution, mix at room temperature for 20 minutes.
11. Centrifuge at 4000 rpm at 4°C for 10 minutes, and collect and keep the supernatant.
12. Add 2x the final volume of ice cold 100% ethanol, mix gently, elute the DNA;
13. Remove excess ethanol, dry DNA sample for five min and re-suspend in 0.1X TE (~300-500 μL).

APPENDIX THREE

Protocol for DNA preparation and amplification for microsatellite marker genotyping

This protocol is for DNA amplification and preparation for microsatellite fragment analysis on the ABI 377 and 3730 instruments.

1. After extraction and quantifying DNA samples make 2.5-5.0 ng/µl dilutions.
2. Aliquot 1 µl of each DNA sample into a 96-PCR plate.
3. The following Table shows the amounts of each ingredient used to prepare a PCR master mix for multiplexing 2 loci at the same time.

Ingredient	1 reaction	100 reactions
Sterile H_2O	3.5 µl	350 µl
10X buffer	0.75µl	75 µl
25mM $MgCl_2$	0.6µl	60 µl
2.5mM dNTP's	0.6µl	60 µl
Primer 1 Reverse (5 µM)	0.5µl	50 µl
Primer 1 Forward (5 µM)	0.5µl	50 µl
Primer 2 Reverse (5 µM)	0.5µl	50 µl
Primer 2 Forward (5 µM)	0.5µl	50 µl
Enzyme AmpliTaq Gold (5U/µl)	0.05µl	5 µl

4. The Table below shows the amount of ingredients used for a singleplex PCR (amplifying 1 locus) reaction.

Ingredient	1 reaction	100 reactions
Sterile H_2O	4.5 µl	450 µl
10X PCR buffer	0.75 µl	75 µl
25 mM $MgCl_2$	0.6 µl	60 µl
2.5 mM dNTPs	0.6 µl	60 µl
Primer Reverse…(5 µM)	0.5 µl	50 µl
Primer Forward…(5 µM)	0.5 µl	50 µl
Enzyme AmpliTaq Gold (5U/µl)	0.05µl	5 µl

5. Mix all ingredients well by pipetting each sample up-and-down a few times.
6. Aliquot 6.5 µl of Master Mix to each well of your plate.
7. Spin plate at 1500 rpm for 1 min if there are bubbles in wells.
8. Cover plate with tube caps.
9. Run program touch60gold which is to follow.

The following touch-down thermocycler PCR program is used for amplification.

1. 94 °C → 8 min.

2. 94 °C → 30 sec.
   60 °C → 30 sec. **2 CYCLES**
   72 °C → 30 sec.

3. 94 °C → 15 sec.

   60 °C → 15 sec (0.5°C decrease with each cycle from 60°C)
   72 °C → 15 sec. **12 CYCLES,**

4. 94 °C → 15 sec.
54 °C → 15 sec. **30 CYCLES**
72 °C → 15 sec.

5. 72°C → 30 min.

6. 4°C hold

Sample preparation for loading on ABI 3730 Instrument.

1. Mix reagents as shown in the table below. Mix using pipette!

Ingredient	1 reaction	100 reactions	comments
Size Std. 500-LIZ	0.1 µl	10 µl	
Hi-Di Formamide	9.0 µl	900 µl	
Sterile H_2O	0.4 µl	40 µl	

2. Aliquot 9.5 µl of mixture to each well (as needed) of your plate. (Plate that fits ABI 3730)!!!

Diluting and Multi-loading of multiple loci on ABI3730

To 2.5 µl of Hi-Di formamide plate. (There's 2.5 µl of formamide in each well you need).

1. Add 0.5* µl of……………………………PCR product. (*- if you have FAM primers (FAM dye is strong),
2. Add 0.5* µl of…………………………… PCR product. Reducing the volume of
3. Add 0.5* µl of…………………………… PCR product. PCR product should be considered- e.g. 0.3 µl)
4. Mix it well!!!!!!!
5. Add 0.5 µl of this mixture to your Size Std. Plate. In each well you should have 10 µl.
6. Denature 2 min. 95°C. After that place immediately on ice for 2 min. Put plates into ABI 3730.

Sample preparation for loading on ABI377

1. Set up GS-500 (ROX) size standard plate ahead of time. Preferably a day ahead of when you plan to run the gel.

Item	Full Plate
GS-350 (Tamra) size std	43.0ul
Loading Buffer	16.0ul
dd H_2O	16.0ul

3. It may be prudent to make extra size standard as the loading buffer tends to be sticky and you end up with not enough for each well in the plate.
   a. Pipette 0.6ul of the size standard solution into each well of a PCR plate cover plate with parafilm
   b. Spin in TJ-25 centrifuge for 2 minutes @ 2000rpm and cover with tinfoil
   c. Store at 4^0C – will evaporate over a few days time
   d. Or after spinning you may poke holes in parafilm at top of each well, cover with tinfoil and place at 37^0C for 1 hour, or leave on bench over night.
4. Preparation of loading mix:
5. Preparation of loading mix:

Item	Full Plate
Deionized formamide	263.0ul
Loading Buffer	11.0ul

6. Set up PCR product/loading mix samples. (General protocol – You need to check the list to determine if dilution needs to be done. Load 2.5uL of loading mix into each well in PCR plate. Add 1.0uL of each PCR product sample. Spin in TJ-25 centrifuge for 2 minutes @ 2000rpm.
7. Set up PCR product / loading mix / size standard samples by:
   a. From the PCR product / loading mix plate transfer 1.2ul of each PCR/LM sample to the dried GS-500 plate.
   b. Cover with parafilm.
   c. Spin in TJ-25 centrifuge for 2 minutes @ 2000rpm.
   d. Cover with tinfoil.
   e. Let plate sit on bench for at least 1 hour, to allow the size standard to go into solution.

f. Denature samples for 5 minutes @ 95^{0}C.

g. Place samples on ice.

h. Place loading tray on ice.

i. Pipette 0.6ul of each sample into loading tray using digital multi-channel pipette

CAUTION!!!

- Sizes and dyes of your primers (if multiplexed or multi loaded) must be compatible with each other e.g. you can multi load PCR products within the same size range if they are labeled with different dyes. There is only one exception; avoid multi loading HEX (green) and NED (yellow) within the same size range because green dye (HEX) when used with filter set G5 produces yellow pull up which can interfere with NED. You can multi load the same dye PCR product if there's at least 30 bp gap in size between them.

APPENDIX FOUR

Details of markers used in QTL scan

BTA	Marker name	Marker type	Location, cM	Alleles	PIC	Heterozygosity	Allelic Diversity
1	TGLA49	SSR	5.06	7	0.45	0.47	0.47
1	VSWAP1D66017A_scf	SNP	12.00	2	0.37	0.51	0.49
1	VSWAP1E15319A_scf	SNP	13.00	2	0.37	0.54	0.49
1	VSWCP1E8363A_scf	SNP	14.00	2	0.36	0.50	0.48
1	VSWCP1E6631A_scf01	SNP	15.00	2	0.37	0.54	0.50
1	AJ496763-027.SP6-429	SNP	15.40	2	0.37	0.52	0.50
1	VSWAP1D54106A_scf01	SNP	21.00	2	0.38	0.99	0.50
1	BV105557-406-K	SNP	60.20	2	0.37	0.52	0.49
1	SCAFFOLD118127_10627	SNP	77.75	2	0.36	0.54	0.48
1	BM6506	SSR	78.17	9	0.28	0.32	0.30
1	SCAFFOLD130531_6932	SNP	89.69	2	0.37	0.55	0.49
1	SCAFFOLD130531_7174	SNP	104.59	2	0.37	0.55	0.49
1	BM1824	SSR	128.11	4	0.69	0.75	0.74
1	AF440368-602	SNP	139.30	2	0.37	0.52	0.48
1	AJ506786-077.T7-347	SNP	148.20	2	0.31	0.38	0.38
1	BMS922	SSR	154.45	8	0.56	0.70	0.60
1	SCAFFOLD151937_3161	SNP	164.75	2	0.36	0.48	0.48
1	SCAFFOLD135366_9135	SNP	169.66	2	0.36	0.49	0.46
2	TGLA44	SSR	3.86	14	0.77	0.76	0.80
2	VSWCP1D3394A_scf01	SNP	10.47	2	0.30	0.37	0.36
2	SCAFFOLD120200_997	SNP	15.58	2	0.32	0.38	0.40
2	SCAFFOLD106077_1598	SNP	22.87	2	0.37	0.46	0.49
2	VSWDP1D6105A_scf	SNP	24.56	2	0.37	0.53	0.49
2	G73155-69-R	SNP	29.00	2	0.34	0.41	0.43
2	VSWAP1E13196A_scf01	SNP	31.36	2	0.37	0.51	0.49
2	CSSM42	SSR	39.25	12	0.73	0.81	0.76
2	SCAFFOLD145177_7279	SNP	57.94	2	0.33	0.60	0.42
2	ILSTS98	SSR	62.09	11	0.77	0.70	0.79
2	AF440377-177	SNP	75.30	2	0.19	0.22	0.21
2	BMS1866	SSR	90.58	12	0.77	0.78	0.79
2	AJ496786-032.T7-100	SNP	104.39	2	0.33	0.42	0.41
2	AJ496786-032.T7-100	SNP	106.80	2	0.33	0.42	0.41
2	VSWDP1D2320A_scf	SNP	113.36	2	0.37	0.53	0.49
2	BM2113	SSR	118.51	9	0.83	0.87	0.85
2	SCAFFOLD107820_1332	SNP	125.88	2	0.36	0.50	0.47
2	SCAFFOLD126235_9784	SNP	127.10	2	0.37	0.57	0.50
2	SCAFFOLD141534_2435	SNP	133.86	2	0.33	0.41	0.41
3	SCAFFOLD125136_12607	SNP	7.59	2	0.37	0.53	0.49
3	UWCA7	SSR	17.37	7	0.40	0.36	0.45
3	BV104041-120-K	SNP	30.60	2	0.37	0.61	0.50
3	BTCN14808	SNP	49.40	2	0.36	0.51	0.47
3	BM4129	SSR	53.53	9	0.61	0.52	0.65
3	SCAFFOLD125836_5424	SNP	60.62	2	0.33	0.57	0.41
3	SCAFFOLD100165_19305	SNP	63.03	2	0.36	0.44	0.47
3	SCAFFOLD141203_6497	SNP	75.75	2	0.37	0.50	0.50
3	BMS1266	SSR	79.62	7	0.66	0.74	0.70
3	VSWAP1E53840A_scf	SNP	89.23	2	0.35	0.71	0.46

BTA	Marker name	Marker type	Location, cM	Alleles	PIC	Heterozygosity	Allelic Diversity
3	SCAFFOLD148432_3242	SNP	90.96	2	0.37	0.49	0.49
3	SCAFFOLD115003_498	SNP	102.87	2	0.37	0.48	0.49
3	VSWDP1E4062A_scf	SNP	103.36	2	0.32	0.45	0.39
3	VSWCP2E4955A_scf01	SNP	116.67	2	0.33	0.45	0.42
3	IDVGA27	SSR	123.00	3	0.56	0.71	0.64
4	BL1030	SSR	7.17	6	0.67	0.71	0.72
4	SCAFFOLD116028_6113	SNP	18.47	2	0.37	0.48	0.50
4	RM188	SSR	28.38	10	0.76	0.79	0.78
4	VSWCP1D7686A_scf	SNP	34.41	2	0.28	0.36	0.33
4	SCAFFOLD105877_5049	SNP	55.75	2	0.35	0.45	0.44
4	BMS3013	SSR	64.66	7	0.35	0.31	0.38
4	BM1500	SSR	87.79	8	0.46	0.59	0.56
4	RM88	SSR	100.16	10	0.64	0.69	0.68
4	SCAFFOLD151159_1914	SNP	105.06	2	0.34	0.40	0.44
4	VSWAP1D65638A_scf	SNP	109.46	2	0.21	0.25	0.24
4	SCAFFOLD141328_8675	SNP	118.80	2	0.34	0.40	0.44
4	SCAFFOLD10509_13171	SNP	123.25	2	0.35	0.48	0.45
4	AF440365-541	SNP	130.00	2	0.29	0.42	0.35
4	AB070368-528-UASMS2	SNP	131.00	2	0.26	0.33	0.30
4	AB070368-1759-UASMS3	SNP	132.45	2	0.36	0.49	0.48
4	AY138588-252	SNP	134.15	2	0.37	0.53	0.48
4	AY138588-305	SNP	135.00	2	0.37	0.52	0.49
4	VSWAP1D5202A_scf01	SNP	137.50	2	0.37	0.50	0.50
4	VSWDP1E4912A_scf	SNP	201.80	2	0.31	0.50	0.38
5	E64H04-38781	SNP	1.17	2	0.36	0.46	0.48
5	E454D12-38941	SNP	4.98	2	0.37	0.48	0.49
5	BM6026	SSR	6.05	11	0.68	0.72	0.70
5	SCAFFOLD125136_12607	SNP	7.59	2	0.37	0.53	0.49
5	VSWAP1D49974A_scf	SNP	13.01	2	0.34	0.46	0.44
5	E189E10-33483	SNP	15.80	2	0.35	0.44	0.45
5	E249A20-36331	SNP	17.80	2	0.36	0.51	0.47
5	E54B08-34244-2	SNP	20.00	2	0.28	0.36	0.33
5	BV105572-86-R	SNP	21.69	2	0.32	0.41	0.41
5	INRA165	SSR	30.00	7	0.62	0.73	0.68
5	BV104041-120-K	SNP	31.60	2	0.37	0.61	0.50
5	AF017143-198	SNP	34.42	2	0.36	0.51	0.47
5	E327D11-34426-2	SNP	45.50	2	0.34	0.51	0.44
5	CSSM34	SSR	46.60	7	0.67	0.75	0.71
5	SCAFFOLD130216_24898	SNP	47.61	2	0.37	0.51	0.50
5	E254B18-36368-1	SNP	58.30	2	0.36	0.54	0.47
5	E261H16-33733	SNP	59.60	2	0.36	0.48	0.47

BTA	Marker name	Marker type	Location, cM	Alleles	PIC	Heterozygosity	Allelic Diversity
5	SCAFFOLD125836_5424	SNP	60.62	2	0.33	0.57	0.41
5	SCAFFOLD100165_19305	SNP	63.03	2	0.36	0.44	0.47
5	SCAFFOLD125222_757	SNP	64.32	2	0.33	0.41	0.41
5	AF440372-229	SNP	65.10	2	0.37	0.52	0.50
5	VSWHP1D1101A_scf01	SNP	71.37	2	0.35	0.52	0.46
5	E193A21-38795-2	SNP	74.20	2	0.37	0.51	0.50
5	SCAFFOLD141203_6497	SNP	75.75	2	0.37	0.50	0.50
5	BM1819	SSR	84.64	6	0.57	0.59	0.61
5	SCAFFOLD148432_3242	SNP	90.96	2	0.37	0.49	0.49
5	E189E10-33481	SNP	95.86	2	0.37	0.48	0.50
5	SCAFFOLD144700_3432	SNP	102.87	2	0.30	0.41	0.37
5	SCAFFOLD115003_498	SNP	104.87	2	0.37	0.48	0.49
5	BV105396-56-Y	SNP	115.45	2	0.35	0.47	0.46
5	AC137534-32977-2	SNP	116.90	2	0.35	0.46	0.45
5	VSWAP1E83749A_scf	SNP	127.17	2	0.37	0.53	0.50
5	BV105351-426-M	SNP	129.77	2	0.37	0.48	0.50
5	BMS597	SSR	131.13	7	0.45	0.50	0.55
6	ILSTS93	SSR	0.00	11	0.67	0.73	0.71
6	SCAFFOLD106320_3036	SNP	6.69	2	0.35	0.47	0.45
6	VSWAP1D84287A_scf01	SNP	20.25	2	0.37	0.49	0.49
6	SCAFFOLD125267_26732	SNP	33.09	2	0.34	0.49	0.44
6	VSWAP1D36890A_scf01	SNP	34.04	2	0.34	0.42	0.43
6	VSWAP1D57857A_scf01	SNP	37.62	2	0.29	0.37	0.36
6	VSWCP1D6874A_scf	SNP	44.49	2	0.37	0.46	0.50
6	VSWAP1D62825A_scf01	SNP	45.10	2	0.36	0.48	0.47
6	BMS382	SSR	52.01	7	0.50	0.51	0.55
6	SCAFFOLD140203_3711	SNP	53.94	2	0.10	0.12	0.11
6	AJ496635-013.SP6-189	SNP	57.50	2	0.37	0.55	0.48
6	AJ496635-013.SP6-280	SNP	57.50	2	0.37	0.56	0.48
6	AJ496635-013.SP6-333	SNP	57.50	2	0.37	0.55	0.48
6	SCAFFOLD15013_21643	SNP	65.93	2	0.31	0.43	0.39
6	VSWAP1D23339A_scf01	SNP	68.51	2	0.37	0.51	0.49
6	RM28	SSR	80.91	4	0.39	0.43	0.45
6	AF061521-1028	SNP	89.70	2	0.31	0.39	0.38
6	SCAFFOLD154819_5764	SNP	95.66	2	0.33	0.42	0.42
6	AF061521-1101	SNP	96.90	2	0.31	0.39	0.39
6	SCAFFOLD111628_3947	SNP	115.22	2	0.37	0.53	0.49
6	BM2320	SSR	130.04	9	0.63	0.70	0.67
7	VSWAP1D50788A_scf01	SNP	4.05	2	0.37	0.50	0.50
7	RM12	SSR	9.13	3	0.18	0.12	0.20
7	VSWAP1D5261A_scf01	SNP	22.49	2	0.37	0.51	0.48

BTA	Marker name	Marker type	Location, cM	Alleles	PIC	Heterozygosity	Allelic Diversity
7	SCAFFOLD110047_28436	SNP	41.90	2	0.29	0.36	0.35
7	TGLA303	SSR	42.45	12	0.84	0.88	0.86
7	AF331034-624	SNP	72.80	2	0.29	0.44	0.36
7	VSWAP1E80685A_scf01	SNP	84.94	2	0.37	0.55	0.50
7	SCAFFOLD10028_2365	SNP	88.08	2	0.35	0.49	0.45
7	BM1853	SSR	89.56	4	0.46	0.27	0.51
7	SCAFFOLD115418_11162	SNP	101.54	2	0.38	0.50	0.50
7	SCAFFOLD145666_13248	SNP	112.22	2	0.37	0.49	0.49
7	SCAFFOLD145666_13245	SNP	112.88	2	0.37	0.48	0.49
7	BTCN16547-2	SNP	120.50	2	0.31	0.39	0.38
7	BTCN16547-4	SNP	120.50	2	0.34	0.51	0.44
7	INRA53	SSR	129.26	12	0.52	0.55	0.56
7	VSWAP1D10129A_scf01	SNP	131.12	2	0.24	0.31	0.28
7	SCAFFOLD15514_11206	SNP	133.56	2	0.37	0.47	0.49
7	SCAFFOLD11377_3357	SNP	138.66	2	0.37	0.55	0.50
8	SCAFFOLD145007_39103	SNP	1.00	2	0.37	0.56	0.50
8	VSWHP1E1323A_scf	SNP	2.54	2	0.37	0.51	0.49
8	BMS1864	SSR	2.69	9	0.58	0.63	0.62
8	SCAFFOLD144544_2876	SNP	10.93	2	0.32	0.45	0.40
8	VSWAP1E38933A_scf	SNP	24.70	2	0.37	0.49	0.50
8	TGLA10	SSR	33.92	4	0.18	0.17	0.20
8	VSWAP1E12741A_scf	SNP	42.94	2	0.37	0.50	0.50
8	VSWAP1E30228A_scf01	SNP	46.74	2	0.37	0.54	0.49
8	VSWAP1D59864A_scf03	SNP	51.59	2	0.31	0.41	0.39
8	VSWAP1D32667A_scf	SNP	63.72	2	0.36	0.50	0.47
8	VSWCP1D2135A_scf01	SNP	67.77	2	0.36	0.49	0.47
8	SCAFFOLD125499_6290	SNP	80.22	2	0.23	0.28	0.27
8	SCAFFOLD120208_7951	SNP	93.79	2	0.37	0.53	0.49
8	BM711	SSR	94.68	8	0.69	0.76	0.73
8	VSWAP1D65476A_scf01	SNP	104.34	2	0.36	0.50	0.48
8	AY297040-1656	SNP	118.70	2	0.36	0.49	0.48
8	CSSM47	SSR	120.87	11	0.38	0.38	0.39
9	ETH225	SSR	12.76	9	0.71	0.79	0.75
9	SCAFFOLD1075_11860	SNP	16.78	2	0.37	0.51	0.49
9	SCAFFOLD110752_5035	SNP	20.00	2	0.35	0.50	0.46
9	AF440371-949	SNP	47.10	2	0.24	0.27	0.28
9	VSWAP1D80085A_scf	SNP	52.17	2	0.37	0.51	0.49
9	SCAFFOLD110029_13524	SNP	62.52	2	0.37	0.52	0.49
9	SCAFFOLD150952_4025	SNP	75.16	2	0.34	0.64	0.44
9	TGLA73	SSR	78.14	9	0.60	0.64	0.66
9	VSWAP1D69443A_scf	SNP	81.01	2	0.37	0.48	0.48

BTA	Marker name	Marker type	Location, cM	Alleles	PIC	Heterozygosity	Allelic Diversity
9	VSWAP1D69443A_scf	SNP	81.01	2	0.37	0.48	0.48
9	SCAFFOLD145441_3461	SNP	86.43	2	0.37	0.53	0.50
9	SCAFFOLD145441_1127	SNP	88.97	2	0.37	0.53	0.50
9	BM3215	SSR	102.44	6	0.56	0.61	0.59
10	AJ496782-118.T7-311	SNP	7.32	2	0.35	0.47	0.46
10	BM6418	SSR	14.30	9	0.62	0.71	0.67
10	AJ245969-1143-Y	SNP	27.10	2	0.37	0.55	0.49
10	AJ245969-750-M	SNP	32.70	2	0.29	0.45	0.35
10	SCAFFOLD10023_31672	SNP	33.55	2	0.37	0.56	0.50
10	SCAFFOLD106386_875	SNP	42.43	2	0.29	0.36	0.36
10	SCAFFOLD106386_542	SNP	43.35	2	0.35	0.47	0.45
10	SCAFFOLD135062_12754	SNP	48.19	2	0.37	0.54	0.50
10	VSWAP1D64403A_scf	SNP	51.99	2	0.37	0.60	0.50
10	VSWAP1E51621A_scf	SNP	54.17	2	0.37	0.53	0.50
10	RM90	SSR	54.99	11	0.50	0.50	0.51
10	SCAFFOLD125539_11512	SNP	55.66	2	0.36	0.50	0.48
10	VSWAP1E61255A_scf	SNP	58.15	2	0.37	0.54	0.50
10	SCAFFOLD11903_9629	SNP	60.68	2	0.37	0.51	0.50
10	SCAFFOLD125279_19173	SNP	69.38	2	0.37	0.57	0.50
10	CSRM60	SSR	78.42	16	0.87	0.75	0.88
10	VSWHP1D1067A_scf	SNP	81.05	2	0.29	0.38	0.36
10	VSWAP1E51639A_scf	SNP	84.02	2	0.37	0.51	0.50
10	SCAFFOLD115093_14103	SNP	87.92	2	0.32	0.42	0.40
10	BV104999-587-Y	SNP	91.21	2	0.37	0.53	0.50
10	VSWAP1D76887A_scf	SNP	102.84	2	0.37	0.55	0.50
10	CSSM46	SSR	105.20	11	0.78	0.74	0.80
11	4013-295-Y	SNP	1.49	2	0.27	0.31	0.32
11	SCAFFOLD140359_23711	SNP	2.58	2	0.37	0.56	0.50
11	SCAFFOLD135323_1575	SNP	7.71	2	0.37	0.50	0.49
11	VSWCP1D6194A_scf01	SNP	9.74	2	0.38	0.57	0.50
11	VSWCP1D1786A_scf01	SNP	9.83	2	0.37	0.55	0.50
11	BM716	SSR	20.05	9	0.58	0.64	0.63
11	SCAFFOLD140043_24985	SNP	22.56	2	0.35	0.47	0.45
11	VSWAP1D11226A_scf	SNP	27.99	2	0.37	0.51	0.50
11	RM96	SSR	41.75	8	0.64	0.71	0.69
11	SCAFFOLD11677_746	SNP	53.13	2	0.37	0.72	0.49
11	SCAFFOLD155239_14627	SNP	59.15	2	0.37	0.53	0.49
11	SCAFFOLD116204_366	SNP	63.87	2	0.33	0.41	0.42
11	SCAFFOLD1537_522	SNP	66.00	2	0.36	0.49	0.48
11	VSWAP1E52580A_scf01	SNP	72.54	2	0.37	0.49	0.50
11	VSWAP1D64514A_scf	SNP	77.67	2	0.37	0.50	0.50

BTA	Marker name	Marker type	Location, cM	Alleles	PIC	Heterozygosity	Allelic Diversity
11	4015-325-R	SNP	86.00	2	0.33	0.42	0.42
11	SCAFFOLD115412_13015	SNP	101.85	2	0.37	0.56	0.49
11	SCAFFOLD125360_791	SNP	106.89	2	0.33	0.43	0.41
11	SCAFFOLD110374_786	SNP	112.40	2	0.37	0.52	0.50
11	VSWAP1D31864A_scf01	SNP	118.75	2	0.37	0.60	0.48
11	HEL13	SSR	123.63	8	0.42	0.47	0.46
12	VSWCP1D6862A_scf01	SNP	4.75	2	0.35	0.47	0.44
12	BMS2057	SSR	20.87	10	0.81	0.82	0.83
12	SCAFFOLD100457_11884	SNP	44.94	2	0.28	0.39	0.34
12	VSWAP1D29337A_scf	SNP	49.37	2	0.37	0.54	0.50
12	BM860	SSR	50.41	9	0.74	0.76	0.77
12	VSWAP1D22911A_scf	SNP	55.96	2	0.26	0.32	0.31
12	SCAFFOLD115574_15191	SNP	63.00	2	0.33	0.42	0.42
12	VSWAP1E54214A_scf03	SNP	67.17	2	0.32	0.45	0.41
12	VSWAP1D24629A_scf	SNP	77.29	2	0.37	0.52	0.49
12	SCAFFOLD125326_1171	SNP	79.48	2	0.36	0.44	0.46
12	RM113	SSR	81.41	6	0.62	0.67	0.68
12	SCAFFOLD130379_11660	SNP	92.46	2	0.33	0.48	0.42
12	INRA209	SSR	109.98	7	0.58	0.70	0.64
13	SCAFFOLD140462_11319	SNP	4.39	2	0.33	0.43	0.42
13	TGLA23	SSR	8.99	13	0.57	0.60	0.59
13	SCAFFOLD150225_6727	SNP	22.94	2	0.36	0.47	0.46
13	SCAFFOLD120395_9107	SNP	24.42	2	0.36	0.48	0.47
13	VSWAP1D21114A_scf01	SNP	33.57	2	0.38	1.00	0.50
13	SCAFFOLD130253_23585	SNP	34.01	2	0.37	0.52	0.50
13	SCAFFOLD1473_926	SNP	41.62	2	0.34	0.49	0.44
13	ILSTS86	SSR	54.65	10	0.71	0.64	0.75
13	VSWAP1E46581A_scf01	SNP	60.13	2	0.34	0.47	0.43
13	SCAFFOLD106976_19933	SNP	67.57	2	0.31	0.41	0.39
13	VSWAP1D13491A_scf02	SNP	79.36	2	0.33	0.45	0.42
13	VSWCP1D5892A_scf01	SNP	80.67	2	0.30	0.36	0.37
13	VSWAP1E18591A_scf	SNP	88.58	2	0.36	0.49	0.48
14	LOC51059	SNP	2.79	2	0.36	0.49	0.47
14	BMS1747	SSR	10.50	11	0.74	0.80	0.77
14	SCAFFOLD105570_18245	SNP	17.05	2	0.31	0.38	0.39
14	SCAFFOLD135027_42531	SNP	17.72	2	0.37	0.49	0.49
14	SCAFFOLD100871_5973	SNP	27.41	2	0.36	0.56	0.48
14	SCAFFOLD134924_5249	SNP	29.15	2	0.36	0.49	0.48
14	BMS1941	SSR	41.71	9	0.63	0.72	0.66
14	SCAFFOLD155146_6074	SNP	43.27	2	0.37	0.52	0.49
14	SCAFFOLD15358_17120	SNP	45.68	2	0.25	0.30	0.29

BTA	Marker name	Marker type	Location, cM	Alleles	PIC	Heterozygosity	Allelic Diversity
14	BL1029	SSR	59.44	12	0.73	0.80	0.76
14	BMS947	SSR	69.79	14	0.80	0.85	0.82
14	SCAFFOLD106433_368	SNP	72.79	2	0.31	0.39	0.38
14	VSWAP1E68252A_scf	SNP	79.88	2	0.32	0.42	0.40
14	VSWAP1E54435A_scf	SNP	90.90	2	0.37	0.51	0.50
14	BL1036	SSR	100.02	11	0.68	0.71	0.71
14	SCAFFOLD15134_29247	SNP	104.03	2	0.37	0.59	0.50
14	VSWHP1E1312A_scf01	SNP	107.15	2	0.36	0.48	0.46
15	MGTG13B	SSR	8.25	5	0.39	0.37	0.41
15	SCAFFOLD120672_10906	SNP	11.90	2	0.26	0.30	0.31
15	VSWAP1E0611A_scf01	SNP	22.00	2	0.35	0.45	0.45
15	VSWAP1E13265A_scf01	SNP	25.42	2	0.38	0.52	0.50
15	SCAFFOLD120241_12465	SNP	25.99	2	0.37	0.55	0.50
15	VSWAP1D50167A_scf01	SNP	29.97	2	0.37	0.49	0.49
15	BV104039-238-S	SNP	31.00	2	0.34	0.46	0.44
15	VSWHP1E0868A_scf01	SNP	40.56	2	0.28	0.35	0.34
15	INRA50	SSR	41.24	10	0.70	0.72	0.73
15	VSWAP1D35274A_scf01	SNP	49.89	2	0.31	0.40	0.39
15	VSWAP1D11046A_scf01	SNP	58.66	2	0.35	0.49	0.44
15	SCAFFOLD136998_2632	SNP	62.08	2	0.35	0.47	0.45
15	AF127030-1099	SNP	64.00	2	0.27	0.32	0.32
15	INRA145	SSR	69.04	7	0.68	0.77	0.72
15	VSWDP1E0785A_scf	SNP	84.12	2	0.37	0.54	0.49
15	VSWAP1D76788A_scf	SNP	96.77	2	0.35	0.46	0.46
15	SCAFFOLD150141_11761	SNP	101.21	2	0.37	0.50	0.50
15	VSWAP1D0723A_scf01	SNP	111.12	2	0.12	0.12	0.13
15	BMS429	SSR	111.14	11	0.80	0.78	0.82
15	VSWDP1E3105A_scf01	SNP	115.97	2	0.37	0.49	0.49
15	SCAFFOLD100290_3465	SNP	118.98	2	0.32	0.42	0.39
16	SCAFFOLD150038_28634	SNP	1.44	2	0.35	0.45	0.45
16	AJ505159-BULGE105-192	SNP	7.12	2	0.30	0.39	0.36
16	VSWAP2E2374A_scf	SNP	12.29	2	0.34	0.41	0.43
16	SCAFFOLD126593_2880	SNP	12.86	2	0.28	0.35	0.34
16	VSWAP1D28783A_scf01	SNP	22.48	2	0.37	0.49	0.48
16	TGLA53	SSR	38.56	12	0.80	0.55	0.82
16	SCAFFOLD145525_4841	SNP	46.56	2	0.37	0.52	0.50
16	SCAFFOLD111164_4522	SNP	64.48	2	0.37	0.49	0.50
16	VSWAP1E7711A_scf01	SNP	65.17	2	0.36	0.49	0.47
16	SCAFFOLD15298_5461	SNP	71.07	2	0.34	0.48	0.44
16	SCAFFOLD102735_1164	SNP	87.55	2	0.36	0.54	0.47
16	VSWAP1E36488A_scf02	SNP	94.49	2	0.32	0.41	0.39

BTA	Marker name	Marker type	Location, cM	Alleles	PIC	Heterozygosity	Allelic Diversity
17	RM156	SSR	2.41	11	0.79	0.68	0.81
17	SCAFFOLD125799_2719	SNP	8.96	2	0.37	0.54	0.50
17	AY147818S1-42T	SNP	9.30	2	0.36	0.72	0.46
17	SCAFFOLD125799_3125	SNP	11.56	2	0.37	0.54	0.50
17	SCAFFOLD125799_3052	SNP	11.78	2	0.37	0.54	0.50
17	VSWCP1D5080A_scf02	SNP	13.32	2	0.37	0.51	0.49
17	VSWCP1E5183A_scf01	SNP	25.65	2	0.37	0.49	0.50
17	SCAFFOLD136321_1119	SNP	51.35	2	0.35	0.51	0.45
17	SCAFFOLD136321_1198	SNP	52.14	2	0.35	0.51	0.45
17	INRA110	SSR	53.51	6	0.43	0.56	0.53
17	CSSM33	SSR	67.91	6	0.58	0.64	0.64
17	SCAFFOLD150220_9590	SNP	73.21	2	0.36	0.49	0.46
17	SCAFFOLD121139_3191	SNP	80.18	2	0.33	0.42	0.42
17	BM1862	SSR	82.02	10	0.70	0.77	0.74
17	SCAFFOLD132028_9406	SNP	82.05	2	0.36	0.49	0.47
17	VSWAP1E41240A_scf01	SNP	86.71	2	0.33	0.44	0.42
17	VSWAP1D44688A_scf	SNP	88.43	2	0.37	0.50	0.50
18	VSWAP1E67989A_scf01	SNP	4.52	2	0.37	0.55	0.50
18	SCAFFOLD110018_29027	SNP	6.76	2	0.37	0.51	0.49
18	BR4206	SSR	8.66	7	0.67	0.59	0.71
18	SCAFFOLD10898_5735	SNP	10.43	2	0.32	0.40	0.40
18	SCAFFOLD149767_1770	SNP	29.05	2	0.37	0.50	0.49
18	INRA121	SSR	30.27	11	0.62	0.68	0.65
18	SCAFFOLD105248_2095	SNP	33.30	2	0.36	0.48	0.47
18	SCAFFOLD135093_34368	SNP	35.17	2	0.37	0.47	0.48
18	AY147817-103	SNP	38.00	2	0.36	0.38	0.47
18	VSWAP1D2872A_scf01	SNP	45.02	2	0.37	0.51	0.50
18	BMS2639	SSR	56.71	13	0.75	0.81	0.78
18	AJ496772-060.SP6-52	SNP	57.90	2	0.37	0.55	0.49
18	BV105445-237-S	SNP	61.00	2	0.37	0.48	0.48
18	BV105352-732-R	SNP	61.68	2	0.37	0.50	0.50
18	SCAFFOLD140034_9410	SNP	65.17	2	0.37	0.48	0.49
18	TGLA227	SSR	87.48	11	0.85	0.86	0.86
19	G73111-168-W	SNP	14.26	2	0.25	0.32	0.30
19	VSWAP1D33690A_scf	SNP	16.22	2	0.35	0.48	0.45
19	X82261	SSR	18.80	9	0.51	0.58	0.57
19	VSWHP1D0986A_scf01	SNP	35.55	2	0.35	0.47	0.44
19	BV105464-366-R	SNP	42.53	2	0.37	0.51	0.49
19	G73114-264-R	SNP	43.05	2	0.37	0.55	0.49
19	AJ496765-039.T7-31	SNP	43.30	2	0.18	0.23	0.20
19	G73133-156-K	SNP	44.00	2	0.37	0.88	0.49

BTA	Marker name	Marker type	Location, cM	Alleles	PIC	Heterozygosity	Allelic Diversity
19	G73160-534-Y	SNP	45.00	2	0.34	0.46	0.44
19	VSWCP1D9335A_scf01	SNP	47.80	2	0.35	0.43	0.46
19	VSWAP1E52060A_scf	SNP	60.56	2	0.35	0.47	0.45
19	IOBT34	SSR	81.10	12	0.65	0.58	0.69
19	BV104969-281-W	SNP	107.90	2	0.37	0.44	0.48
19	BMS601	SSR	111.59	8	0.59	0.53	0.63
20	VSWAP1E51738A_scf01	SNP	15.45	2	0.37	0.51	0.50
20	CART-SNP	SNP	16.87	2	0.37	0.50	0.50
20	SCAFFOLD100002_29562	SNP	22.26	2	0.31	0.40	0.39
20	SCAFFOLD127505_336	SNP	30.19	2	0.33	0.46	0.42
20	TGLA126	SSR	31.93	8	0.52	0.61	0.60
20	SCAFFOLD108770_1519	SNP	44.55	2	0.31	0.40	0.39
20	AF140284-200	SNP	59.00	2	0.31	0.38	0.38
20	SCAFFOLD15454_18507	SNP	59.15	2	0.36	0.51	0.48
20	VSWAP1D32989A_scf03	SNP	61.42	2	0.35	0.43	0.45
20	VSWAP1D51959A_scf01	SNP	61.50	2	0.37	0.52	0.48
20	SCAFFOLD150139_6440	SNP	72.32	2	0.37	0.46	0.49
20	AJ496641-007.SP6-271	SNP	72.50	2	0.30	0.33	0.36
20	SCAFFOLD132982_3281	SNP	74.30	2	0.36	0.48	0.47
20	VSWAP1E44737A_scf	SNP	77.20	2	0.37	0.50	0.49
20	UWCA26	SSR	77.71	12	0.56	0.58	0.58
21	BM8115	SSR	0.00	10	0.52	0.52	0.54
21	VSWAP1D34821A_scf	SNP	1.03	2	0.37	0.51	0.49
21	AJ496767-048.SP6-638	SNP	16.85	2	0.36	0.46	0.47
21	SCAFFOLD155320_17634	SNP	22.42	2	0.37	0.54	0.49
21	ILSTS95	SSR	24.40	7	0.44	0.53	0.50
21	SCAFFOLD141275_7008	SNP	28.76	2	0.36	0.49	0.47
21	SCAFFOLD141409_4380	SNP	29.28	2	0.37	0.52	0.50
21	AJ496767-048.SP6-493	SNP	29.40	2	0.37	0.51	0.49
21	AJ496767-048.SP6-638	SNP	29.40	2	0.35	0.51	0.46
21	SCAFFOLD126032_4723	SNP	32.92	2	0.37	0.54	0.50
21	SCAFFOLD121099_3123	SNP	36.14	2	0.37	0.51	0.50
21	SCAFFOLD153044_4148	SNP	43.61	2	0.31	0.40	0.38
21	VSWAP1D81486A_scf01	SNP	49.48	2	0.36	0.47	0.47
21	TGLA337	SSR	56.10	7	0.54	0.69	0.62
21	2279-422-W	SNP	57.90	2	0.35	0.47	0.46
21	2279-422-W	SNP	65.20	2	0.35	0.46	0.45
21	VSWAP1D62714A_scf	SNP	70.32	2	0.29	0.35	0.35
21	BMS670	SSR	81.50	3	0.11	0.06	0.12
22	SCAFFOLD135781_9137	SNP	11.70	2	0.34	0.42	0.43
22	BV104328-247-R	SNP	17.99	2	0.05	0.05	0.05

BTA	Marker name	Marker type	Location, cM	Alleles	PIC	Heterozygosity	Allelic Diversity
22	HUJ175	SSR	31.50	8	0.32	0.35	0.33
22	SCAFFOLD135112_7636	SNP	40.51	2	0.37	0.52	0.50
22	VSWDP1E6267A_scf	SNP	40.64	2	0.33	0.45	0.42
22	VSWAP1E50506A_scf01	SNP	45.71	2	0.37	0.54	0.50
22	CSSM41	SSR	46.30	6	0.46	0.51	0.55
22	VSWAP1D52487A_scf01	SNP	46.78	2	0.37	0.50	0.49
22	VSWAP1E28213A_scf01	SNP	60.44	2	0.36	0.48	0.47
22	VSWAP1E80014A_scf01	SNP	65.21	2	0.33	0.42	0.42
22	VSWAP1D0860A_scf	SNP	71.86	2	0.35	0.49	0.44
22	VSWAP1D58658A_scf	SNP	72.99	2	0.30	0.35	0.36
22	G73143-166-R	SNP	74.00	2	0.10	0.11	0.10
22	VSWAP1E65803A_scf	SNP	74.93	2	0.36	0.48	0.48
22	HMH1R	SSR	77.00	6	0.46	0.50	0.51
22	VSWCP1D3545A_scf	SNP	82.72	2	0.35	0.48	0.46
22	SCAFFOLD115037_42701	SNP	91.99	2	0.29	0.45	0.35
22	VSWAP1E56425A_scf01	SNP	96.26	2	0.37	0.54	0.50
23	3945-274-Y	SNP	0.00	2	0.33	0.40	0.42
23	INRA132	SSR	0.00	5	0.53	0.50	0.61
23	3915-125-S	SNP	9.00	2	0.28	0.38	0.34
23	SCAFFOLD102055_10178	SNP	9.22	2	0.36	0.49	0.48
23	VSWAP1E34839A_scf01	SNP	9.72	2	0.36	0.46	0.47
23	VSWAP1E15517A_scf02	SNP	13.95	2	0.36	0.53	0.47
23	2561-253-R	SNP	15.00	2	0.13	0.15	0.14
23	VSWAP2D2362A_scf02	SNP	21.87	2	0.32	0.40	0.39
23	AJ505161-BULGE128-92	SNP	24.20	2	0.37	0.54	0.50
23	SCAFFOLD105090_27305	SNP	35.37	2	0.37	0.51	0.49
23	BOLA-DRB1	SSR	35.40	12	0.82	0.84	0.84
23	G73136-248-R	SNP	38.00	2	0.37	0.55	0.50
23	VSWAP1D22330A_scf	SNP	45.78	2	0.31	0.39	0.39
23	VSWAP1D80993A_scf02	SNP	48.13	2	0.31	0.39	0.38
23	G73136-248-R	SNP	56.42	2	0.37	0.55	0.50
23	CSSM24	SSR	58.40	20	0.80	0.80	0.82
23	VSWAP1D51116A_scf	SNP	61.19	2	0.37	0.52	0.50
23	VSWAP1E52156A_scf01	SNP	64.96	2	0.34	0.50	0.43
23	VSWAP1E65704A_scf	SNP	67.36	2	0.32	0.40	0.40
23	VSWAP1E41860A_scf02	SNP	72.15	2	0.36	0.46	0.47
23	VSWAP1D36867A_scf	SNP	78.72	2	0.38	0.52	0.50
23	SCAFFOLD155467_15287	SNP	88.57	2	0.37	0.49	0.48
24	BM226	SSR	6.00	2	0.44	0.48	0.46
24	VSWAP1D0445A_scf01	SNP	19.21	2	0.22	0.27	0.25
24	VSWAP1E52926A_scf01	SNP	19.88	2	0.37	0.39	0.50

BTA	Marker name	Marker type	Location, cM	Alleles	PIC	Heterozygosity	Allelic Diversity
24	VSWAP1D50358A_scf	SNP	21.26	2	0.35	0.42	0.44
24	AJ505157-BULGE100-78	SNP	26.44	2	0.37	0.50	0.50
24	ILSTS101	SSR	34.40	5	0.49	0.55	0.54
24	VSWAP1E2485A_scf01	SNP	36.90	2	0.36	0.49	0.47
24	SCAFFOLD105690_6594	SNP	42.10	2	0.31	0.39	0.39
24	VSWAP1D59386A_scf	SNP	45.08	2	0.34	0.40	0.43
24	BMS3024	SSR	62.50	3	0.53	0.60	0.57
24	VSWAP1E32801A_scf	SNP	63.25	2	0.38	0.55	0.50
24	MC4R-AF265221-1069	SNP	67.42	2	0.37	0.47	0.49
24	VSWAP1D38233A_scf	SNP	69.41	2	0.32	0.40	0.40
24	VSWAP1D23311A_scf01	SNP	73.48	2	0.36	0.49	0.46
25	SCAFFOLD146466_8003	SNP	14.74	2	0.37	0.49	0.48
25	SCAFFOLD105457_745	SNP	14.94	2	0.37	0.50	0.49
25	ILSTS46	SSR	29.70	10	0.72	0.60	0.76
25	VSWCP1E8344A_scf	SNP	30.74	2	0.20	0.25	0.23
25	VSWAP1D24995A_scf	SNP	46.35	2	0.33	0.41	0.42
25	VSWAP1D28027A_scf	SNP	50.93	2	0.33	0.43	0.42
25	SCAFFOLD10375_2467	SNP	58.09	2	0.34	0.43	0.43
25	AF5	SSR	60.60	12	0.76	0.80	0.77
25	SCAFFOLD100231_11024	SNP	61.76	2	0.27	0.35	0.32
25	SCAFFOLD100231_10975	SNP	61.99	2	0.37	0.52	0.50
25	SCAFFOLD100231_10874	SNP	62.44	2	0.37	0.52	0.50
26	SCAFFOLD109398_2406	SNP	11.77	2	0.37	0.51	0.49
26	SCAFFOLD110120_25106	SNP	15.27	2	0.31	0.36	0.38
26	SCAFFOLD152289_8581	SNP	34.41	2	0.38	1.00	0.50
26	RM26	SSR	37.30	8	0.79	0.79	0.81
26	G73121ML3	SNP	44.00	2	0.37	0.53	0.50
26	BV105383-115-R	SNP	46.06	2	0.37	0.58	0.50
26	BM804	SSR	59.60	8	0.69	0.74	0.74
26	SCAFFOLD130770_5013	SNP	69.22	2	0.25	0.30	0.29
27	BM871	SSR	7.70	12	0.53	0.50	0.55
27	SCAFFOLD10151_22515	SNP	11.19	2	0.33	0.44	0.41
27	SCAFFOLD105280_12649	SNP	25.36	2	0.38	0.48	0.50
27	SCAFFOLD105280_12481	SNP	25.57	2	0.37	0.48	0.50
27	SCAFFOLD105280_12735	SNP	25.57	2	0.38	0.48	0.50
27	VSWAP1D83280A_scf	SNP	31.12	2	0.37	0.50	0.50
27	VSWAP1D72625A_scf	SNP	31.78	2	0.37	0.50	0.49
27	CSSM36	SSR	38.90	9	0.64	0.72	0.69
27	VSWAP1E43769A_scf01	SNP	39.25	2	0.34	0.48	0.44
27	SCAFFOLD127325_1394	SNP	42.28	2	0.33	0.43	0.42
27	SCAFFOLD111293_9353	SNP	52.14	2	0.37	0.52	0.50

BTA	Marker name	Marker type	Location, cM	Alleles	PIC	Heterozygosity	Allelic Diversity
27	SCAFFOLD120838_12637	SNP	55.55	2	0.37	0.50	0.50
27	SCAFFOLD120249_11968	SNP	62.52	2	0.24	0.31	0.29
27	BMS1675	SSR	64.10	10	0.56	0.64	0.62
28	BMS2060	SSR	0.00	6	0.48	0.52	0.53
28	VSWAP2E26079A_scf	SNP	5.08	2	0.37	0.47	0.49
28	SCAFFOLD131537_8388	SNP	11.74	2	0.31	0.37	0.38
28	SCAFFOLD131537_5113	SNP	12.16	2	0.36	0.52	0.48
28	SCAFFOLD127004_3536	SNP	17.01	2	0.34	0.45	0.43
28	BMS510	SSR	22.10	14	0.81	0.85	0.83
28	VSWAP1E0923A_scf	SNP	22.58	2	0.37	0.46	0.49
28	VSWAP1E61890A_scf	SNP	22.68	2	0.37	0.46	0.50
28	VSWAP1D67089A_scf	SNP	24.89	2	0.05	0.05	0.05
28	SCAFFOLD11052_910	SNP	27.51	2	0.37	0.49	0.50
28	SCAFFOLD150989_2786	SNP	32.24	2	0.33	0.41	0.41
28	VSWAP1D50910A_scf02	SNP	35.53	2	0.37	0.49	0.49
28	VSWAP1D69565A_scf01	SNP	35.54	2	0.37	0.54	0.50
28	BMS2200	SSR	43.30	11	0.77	0.79	0.79
28	VSWAP1D51369A_scf	SNP	43.98	2	0.37	0.54	0.50
28	AJ496776-090.T7-421	SNP	47.80	2	0.37	0.53	0.49
28	AJ496776-090.T7-429	SNP	47.80	2	0.37	0.49	0.50
28	VSWAP1D76531A_scf01	SNP	50.04	2	0.37	0.50	0.49
28	SCAFFOLD11756_13885	SNP	51.39	2	0.21	0.22	0.23
28	SCAFFOLD121397_6630	SNP	59.46	2	0.37	0.55	0.48
29	BTCN44447-2	SNP	0.00	2	0.20	0.23	0.23
29	VSWAP1E18955A_scf01	SNP	3.82	2	0.32	0.38	0.39
29	BMS764	SSR	9.70	9	0.79	0.83	0.81
29	BV104046-170-R	SNP	11.29	2	0.37	0.57	0.49
29	VSWAP1E32347A_scf01	SNP	15.33	2	0.19	0.22	0.21
29	RM179	SSR	22.40	5	0.51	0.57	0.59
29	SCAFFOLD145192_9213	SNP	24.87	2	0.36	0.49	0.47
29	VSWAP1D64146A_scf	SNP	32.72	2	0.31	0.39	0.38
29	VSWAP1D67557A_scf	SNP	39.18	2	0.32	0.41	0.40
29	SCAFFOLD141082_7705	SNP	39.91	2	0.36	0.49	0.48
29	3104-421-R	SNP	46.41	2	0.29	0.37	0.36
29	IDVGA7	SSR	50.10	12	0.24	0.22	0.25